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TREATMENT OF HAY-FEVER AND ITS ALLIED CONDITIONS BY IONIZATION. PRELIMINARY REPORT.

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More than ten years ago Demetriades¹ demonstrated in the Vienna Ear, Nose and Throat Clinics, a method for the treatment of hyperesthetic rhinitis which he termed "Iontophoresis." In this treatment the nose was packed with cotton wool saturated with an electrolyte containing calcium salts. Into the midst of the packing was inserted a zinc electrode which, in turn, was connected to the positive pole of a galvanic apparatus. Using from two to three milliamperes of current over a period of from five to ten minutes, he was able to obtain considerable relief in the patient's symptoms. He believed that the positively charged metallic ions which were driven into the nasal mucosa desensitized the nerve endings and made them less susceptible to irritation by allergens.

Franklin,² in England, later used a modification of the same method, but substituted a 1 per cent zinc sulphate solution for the calcium in the electrolyte. He considerably increased the electric dosage, in that he gave from three to five milliamperes for from 15 to 20 minutes. In 1931 he reported a series of 22 hay-fever and three vasomotor rhinitis cases in which he was able to secure permanent cures by this method.

He states that the majority of cases require two ionizations for complete relief of their symptoms and that in no case was there any untoward results from this form of treatment.

For many years I had been dissatisfied with the treatment of hay-fever in vogue in America during this time. At best, the results from the standard methods of therapy are unsatisfactory in many cases and transitory in practically all of them. Eight years ago I became interested in ionization for this condition and for the past seven years have used this method to the exclusion of all other forms of treatment. In my earlier work I used a method similar to that elaborated by Franklin, but gradually increased the amount of current to ten milliamperes and shortened the period of exposure, using one side of the nose at a time for ten minutes. The results of this were much more satisfactory than I had been able to secure from any other form of therapy. The nasal reactions, however, following the earlier treatments were quite severe and in some cases occasioned the patients considerable nasal discomfort for several days following the ionization. In an effort to reduce these reactions, I tried other metals for the electrode and other substances for the electrolyte, and finally found that a combination of zinc, cadmium and tin with their respective salts used as the electrolyte gave better results and much less irritation as a post-operative reaction to the treatment. For the last four years I have used this combination exclusively and in no case has the nasal reaction lasted longer than a few hours following the ionization. The technique which I have found most satisfactory is as follows:

After the patient is admitted my assistant obtains a history of the case in detail and then an examination of the nose is made. If the nasal septum is found to be greatly deviated, I do not attempt an ionization treatment, for it would be impossible to so pack the nose as to completely cover the entire mucous membrane. In such a case a submucous resection is indicated and the patient is advised that an ionization treatment may be given in about six weeks following the operation. Even with a straight septum, it is sometimes difficult to pack the nose so that the postnasal space and the turbinates are entirely covered.

As a preliminary to the ionization treatment, each side of the nasal space is packed with 10 per cent cocaine in 1.5 per cent ephedrine solution for ten minutes. This shrinks and anesthetizes the nasal membrane and greatly minimizes the discomfort which might otherwise be experienced. Cotton strips one-half inch wide and about three feet long, and as thin as can be handled, are saturated with the ionizing solution and packed into the nose as far back as the postnasal space. The electrode is wrapped with a spiral of cotton and also saturated with the solution, then introduced into the nose and thoroughly packed in with more saturated cotton. The electrode is connected with the positive pole, and the negative pole is placed on the right arm and kept wet with saline solution. A special resistance meter is connected with the patient to measure his electrical resistance. If, after the nose has been packed, the resistance between the arm and the nose is as high as 8,000 to 10,000 ohms, it is positive evidence that the nose is not properly packed, and the packing must be withdrawn and replaced until a suitable reduction of resistance occurs. I have found 2,500 to 3,000 ohms to be quite satisfactory. This measurement and adjustment of resistance is an important procedure and I feel sure that a number of my early failures have been due to insufficient or improper packing of the nose in preparation for the ionization.

After the nose is properly packed and the patient's resistance tested, the current is turned on gradually and the patient is watched carefully until the proper milliamperage is obtained, as noted on the milliammeter. Immediately after the current attains its maximum, a metallic taste is noted, there is an increase in the amount of saliva and lacrimation, and a slight flush appears on the face. The strength of the current should never reach the point of coagulation; I have found that 100 milliamperes minutes is a sufficient treatment for one side of the nose.

The type of current is of the utmost importance in ionization therapy, as has been shown by Stevenson³ in his exhaustive analysis of the physics and chemistry of the ionization methods of treatment. It should be a direct current obtained from a small motor generator, capable of delivering at least 30 milliamperes when connected in series with the patient.

In my own machines I have installed a filter on each side of the current to smooth out any ripples that may arise between the brush and the commutator. When one realizes how extremely dangerous it is to use any form of alternating current in such intimate relationship with the brain, as with improvised apparatus, such as a rectifier or rotatory converter, where there is a possibility of an alternating current getting into the patient's circuit, then it is understood why it is necessary that only a ground-free motor generator, with a limit stop, be used. Furthermore, it is highly desirable that this machine be so constructed with interlocking switches and resistance meter that the patient's resistance be measured and correct readings be obtained without shock or any electrical sensation whatsoever to the patient. Several physicians who have observed this work and who had previously constructed electric wall plates or other machines which would put out a suitable amount of direct current, have tried this treatment using these machines as a substitute for the one I have described above. Invariably, the report has been that the patient complained bitterly of the discomfort connected with the treatment and the consciousness all through the ionization of the discharge of electricity into the nose and arm. The results from such machines have been very unsatisfactory, so that I feel that a proper machine, proper electrodes and proper electrolyte is the absolute *sine qua non* for success in this method of treatment. Chemical analysis of the electrode, both before and after the treatment, has revealed the fact that the ten-minute ionic displacement which takes place from the electrode into the tissues during treatment changes the chemical makeup of the electrode so that after usage it should be discarded instead of being used the second time.

After the current has passed for a sufficient length of time, it is gradually turned off and the electrode and packs are carefully removed. The tissues are now seen to be contracted and the turbinates and septum are covered with a gray or white coating. Though, before, the treatment the patient may complain of watery, itching eyes, itching ears and palate and sneezing, these symptoms disappear almost immediately after the ionization treatment.

For the first 12 hours following the ionization, the patient may be uncomfortable and complain of headache, irritation

of the eyes and obstructed nose. As a rule, all patients are able to report to the office the following day. Almost without exception the allergic symptoms are entirely gone and aside from the nasal obstruction, patients are very comfortable. When seen on the day following the treatment the nose is usually entirely obstructed with a heavy white or grayish membrane which clings tightly to the mucosa and usually bridges across from the septum to the lateral wall of the nose. This membrane should not be disturbed until the third day, when, if it is still present, it may be gently loosened by passing a ball-pointed probe between the membrane and the mucosa underlying it. Then it will be easy to pick up the membrane with a pair of forceps and remove it from the nose. Following this, a bland shrinking spray should be used for several days.

The real criterion of efficiency of any allergic treatment is the permanence in the relief of symptoms secured for the patient. Because of this, I am not reporting any of my recent cases, but am including only those cases which have been entirely relieved of their symptoms for more than one year. All of the patients reported here had hay-fever as their major allergic manifestation, but ten of them were complicated with other manifestations of the disease. Four of them suffered from asthma in addition to their hay-fever, and six had perennial hyperesthetic rhinitis. All but one of these patients have been entirely relieved of their symptoms for more than a year, and nineteen of them for more than three years.

Thirty-one in this group of forty patients required only one ionization for complete relief, seven required two ionizations, and in one of them it was necessary to repeat the ionization the third time before permanent relief from symptoms was obtained. One case was entirely relieved of symptoms for one year, then returned and was given two more ionizations without any relief whatsoever. I am entirely at a loss to understand the failure to secure a result in this case. All but eight of these patients had had the sensitization tests and in 14 of those there were other positive reactions present in addition to the autumnal pollens. Several of them had, in addition to the hay-fever, food sensitivities and the majority of these patients after ionization apparently lost their sensitivity to these foods because contact with them no longer produced

Name	Age	Sex	Duration of Disease	Form of Disease	Previous Treatment	Reaction from Ionization	Number of Ionization Trts.	Results as to Recurrence Cases Ionized	Year Trt. Given
J.E.W.	56	F.	20 years	H. F.	No	Severe	1	No recurrence	1927
M.M.	58	F.	10 years	H. F.	No	Severe	1	No recurrence	1927
H.L.W.	53	F.	18 years	H. F.	Nasal resect. & vac.	Severe	1	No recurrence	1930
J.C.Q.	29	F.	5 years	H. F.	Vaccines	Severe	1	No recurrence	1929
M.S.	33	M.	5 years	H. F.	Nasal resection	Mild	3	Hay-fever cured each season but recurred following season	1931-32-33
Z.R.	13	M.	Many yrs. (5?)	H. F. & Hyperesthetic Rhinitis	Vaccines	Moderate	1	No recurrence	1931
W.F.M.	30	M.	Many years	H. F. & Hyperesthetic Rhinitis	No history	Severe	1	No recurrence	1928
W.H.H.	65	M.	Many years	H. F. & Rhinitis	Many local trts.	Severe	1	No recurrence	1929
R.H.	34	M.	10 years	H. F.	Vaccines	Severe	1	No recurrence	1928
M.H.	53	M.	15 years	H. F.	No	Severe	1	No recurrence	1928
E.L.M.	25	F.	5 years	H. F.	Vaccines	Severe	1	No recurrence	1929
G.D.	42	F.	Many years	H. F. & Hyperesthetic Rhinitis	Antral puncture	Severe	1	No recurrence	1929
J.D.D.	47	F.	5 years	Hyperesthetic Rhinitis	Local treatments	Severe	1	No recurrence	1930
N.C.	40	F.	No history	H. F.	Vac. & local trts.	Severe	1	No recurrence	1929
F.L.Q.	15	F.	3 years	Hyperesthetic Rhinitis	Vac. & local trts.	Severe	1	No recurrence	1929
C.O.Q.	18	M.	5 years	H. F.	Vac. & local trts.	Severe	2	Improved 3 years after 1st trt. and cured after 2nd trt.	1930 & 1933
B.R.	36	M.	5 years	H. F.	Local treatments	Moderate	2	2 trts. in 2 wks. gave complete relief; no recurrence	9-4-30 & 9-20-30
N.C.	35	M.	No history	H. F.	Local treatments	Moderate	1	No recurrence	1930
B.Z.	18	M.	5 years	H. F.	None	Moderate	1	No recurrence	1930
J.W.G.	49	M.	15 years	H. F.	Local treatments	Moderate	3	1930 trt. gave complete relief 2 trts. in 1932; no relief	1930(1) 1932(2)
R.L.G.	45	M.	3 years	H. F.	Local treatments	Moderate	1	No recurrence	1931

Name	Age	Sex	Duration of Disease	Form of Disease	Previous Treatment	Reaction from Ionization	Number of Ionizations	Results as to Recurrence Cases Ionized	Year Trt. Given
J.A.C.	50	F.	50 years	H. F.	Local treatments	Moderate	2	1st trt. gave very little relief; 2nd, complete relief	1932(2)
D.L.F.	28	M.	10 years	H. F.	Resect. & loc. trts.	Moderate	2	1st trt. gave complete relief for 1 yr. 2nd trt., 1 yr. ago, also complete relief	1931 & 1932
B.F.	13	M.	2 years	H. F. & Hyperesthetic Rhinitis	None	Moderate	1	No recurrence	1931
H.T.C.	40	M.	3 years	H. F.	Resect septum and local treatments	Moderate	2	1st trt., cured for 2 yrs. 2nd trt., 4 mos. ago and no recurrence	1931 & 1933
V.Z.L.	33	F.	10 years	H. F.	Vaccines	Moderate	1	No recurrence	1932
D.O.L.	48	M.	27 years	H. F. & Asthma	Local treatments	Moderate	1	No recurrence	1932
H.E.M.	49	M.	10 years	H. F.	Local treatments	Moderate	1	No recurrence	1932
H.M.	35	M.	7 years	H. F.	Local treatments	Moderate	1	No recurrence	1931
N.M.	53	F.	11 years	H. F.	Local treatments	Moderate	1	No recurrence	1932
R.H.	34	F.	5 years	H. F.	Local treatments	Moderate	1	Cured for 2 yrs., on 3rd autumnal season two light attacks	1931
E.H.H.	19	M.	4 years	H. F.	Local treatments	Moderate	1	No recurrence	1932
W.H.J.	35	F.	Many years	H. F.	Local treatments	Moderate	1	No recurrence	1932
J.M.W.	43	F.	15 years	H. F.	Local treatments	Moderate	1	No recurrence	1932
J.W.	40	M.	2 years	H. F.	None	Moderate	1	No recurrence	1932
M.K.W.	32	F.	7 years	H. F.	Local treatments	Moderate	2	1st trt., no relief; 2nd trt., 1 mo. later, complete relief	1932
D.C.	39	M.	Many years	H. F. & Asthma	Local treatments	Moderate	2	1st trt. cured H. F. & asthma 1 yr. 2nd trt. 9 mos. ago, no recurrence	1932 & 1933
M.W.	8	M.	8 years	H. F. & Asthma	None	Moderate	1	No recurrence	1932
B.C.	14	M.	5 years	H. F.	None	Slight	1	Trt. 4 mos. ago during his season stopped hay fever, and no recurrence	1933
A.C.E.	24	F.	7 years	H. F.	Local treatments	Slight	1	Trt. 6 mos. ago, cured H. F. and no recurrence	1933

nasal symptoms. Eight of the total group had received no treatment whatsoever for either the hay-fever or the other symptoms, while 32 of them had had all sorts of therapy, including the desensitization by vaccines, local treatment and nasal operations, all without relief from their symptoms.

It is noteworthy that in the first 16 of these patients, which were all then done more than three years ago, the reactions following the ionization were much more severe than in recent years. This is, I am sure, due to the modification which I had made in the electrode and electrolyte. The first group of these cases were done, using the plain zinc electrode, but since the cadmium and tin have been combined with the zinc, the local reactions have been very much less severe than previously.

The exact physiological mechanism involved in the prompt relief of allergic symptoms following ionization of the nose, I am not as yet able to explain. Much work remains to be done before this method of therapy can be placed upon an accurate scientific basis. As yet, it is only empirical, in that careful use has definitely demonstrated that it will relieve the patient's symptoms. Many problems connected with this are being investigated and it is hoped that within the next year or so, a much fuller report may be given which will carry a more adequate explanation of just what chemical change takes place in the patient's body following ionization.

At first I thought that I was producing merely a local change in the patient's nasal mucosa which perhaps rendered the nerve endings less sensitive to allergens, but further work, particularly on certain cases of food allergies, manifested by urticaria and asthma, and cases of angioneurotic edema, who had never complained of nasal symptoms, have convinced me that the reaction is not local, but systemic.

CONCLUSION.

Ionization of the nasal mucosa, using a zinc, cadmium and tin electrode and salts of the above solutions in the electrolyte, produces a chemical change in the patient ionized which renders him less sensitive to substances to which he previously gave definite allergic manifestations.

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Medical Arts Building.

**ALCOHOLIC INJECTIONS INTO THE INFERIOR NASAL
TURBINATES FOR THE AMELIORATION OF
THE SYMPTOMS OF THE COMMON
COLD, ROSE-FEVER, HAY-FEVER,
AND HAY-FEVER ASTHMA.**

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The injection of a 60 per cent to 70 per cent alcoholic solution into a sensory nerve, the nerve sheath, or even along its pathway, if the alcohol is absorbed by the nerve, will produce a more or less transitory or permanent anesthesia of the area supplied by the alcohol injected nerve.

The inferior nasal turbinate bodies are the most sensitive and most responsive areas or organs in the nose. They are the thermostatic bodies or organs which react to the slightest thermal changes resulting in contraction, thereby enlarging the calibre of the nose or in engorgement, thereby occluding the nose. The inferior turbinates exert a biological protective function to the nose, which is analogous with the biological protective function of the eyelids in relation to the eyes.

Excessive or irritating secretions flowing from the nasal accessory sinuses or the tear ducts and coming in contact with the inferior turbinates cause an instantaneous reaction of the inferior turbinates accompanied by the biological reaction of sneezing. The inferior turbinates guard the entrance to the nose and they endeavor to arrest all physical and chemical substances which may irritate the nose, the nasopharynx, the sinuses and the respiratory tracts.

Rose-fever, hay-fever, hay-fever asthma as well as many other allergic reactions, we believe, are due to a definite pollen or some specific substances and the clinical reactions are due to their physical presence, their chemical irritations or their products of decomposition. These substances may enter the nasal respiratory tracts either by way of the nose or possibly

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through the tear ducts. In all cases of irritation the inferior turbinates manifest a reaction by occluding the nares, producing violent sneezing, accompanied by an edema of all of the mucous membranes of the nares and accessory nasal sinuses by a conjunctivitis, lacrimation, rhinorrhea and if the process repeats itself over a period of years, an allergic asthma may develop.

From the experience of administering alcohol injections for the amelioration and relief of neuralgias involving the course of the trifacial nerve, the sphenoid ganglion, the infra orbital and the inferior dental and palatal nerves, by analogy alcohol injected into the body of the inferior turbinates should render these sensitive nasal organs anesthetic and block the irritating impulses resulting from sinus secretions, specific pollens or chemicals. Technique of the alcohol injections into the inferior turbinates: A fine, long hypodermic needle is inserted directly into the body of the inferior turbinate and 0.1 cc. of 2 per cent novocaine with a 1/50000 adrenalin solution is injected. This produces a partial anesthesia and contraction of the turbinate body.

Without removing the needle the hypodermic is detached and a second hypodermic, which is already prepared with 0.5 cc. of 60 per cent to 70 per cent alcohol solution (stronger alcoholic solutions may cause excessive swelling and pressure necrosis of the inferior turbinate body) is attached to the needle which has remained in the body of the inferior turbinate and the alcohol is slowly injected.

The patient usually complains of a sharp burning sensation radiating up towards the eye and over the side of the face and sometimes to the teeth. When the patient is conscious of this radiating pain one can be reasonably sure that the alcohol was injected into the nerve filaments or at least is being absorbed by them.

A successful injection is always followed by very sharp reaction. For two or three days the inferior turbinate is blanched, edematous and fills the entire lower and middle meatus. After the second or third day the edema gradually disappears so that at the end of a week or ten days the turbinate body is firmly contracted over the turbinate bone, then the inferior turbinate in the other nostril is injected. The

degree of anesthesia depends on the strength of the alcohol and the amount of absorption by the nerves.

The period of anesthesia may last from a few days to two or three years. During the time when the inferior turbinates are partially or totally anesthetic, the patient has certain uncanny experiences. They have a partial or total loss of the normal sensations coming from the lower turbinates and often over an area beyond. For a considerable period their nose may drip without their being quite conscious of the same; they cannot feel the air going in or out of the nose as formerly and when they blow their nose it feels different than before the injection. Some patients even state that for a time their cheek feels numb and swollen.

Results: These alcoholic injections into the inferior turbinates have been administered from one week to three months prior to the expected attack, to patients suffering from the classical autumnal head colds, those suffering from rose-fever, hay-fever and other allergic reactions, as well as to patients who suffer from hay-fever asthma. The patients have varied in age from rather young children to the chronic sufferer of middle age. In all cases there has been some amelioration of the patients' irritating symptoms and their extreme nasal discomfort. This may be due only to the temporary or prolonged contraction of the inferior turbinate bodies resulting in a freer nasal ventilation and drainage. Secondly, these results may be due to the anesthetic effect of the inferior turbinates and the blocking of irritating reactions coming to and through these organs. In all cases the persistent sneezing is lessened to a marked degree and the partial control of the sneezing factor lessens most of the other symptoms which are initiated and aggravated by violent and prolonged sneezing. Many of the extreme asthmatic symptoms are lessened. The alcoholic injections into inferior turbinates afford amelioration to patients suffering from the common cold, from rose-fever, hay-fever, hay-fever asthma or other forms of nasal allergic reactions.

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THE COMMON COLD AND ITS TREATMENT WITH HOMIODIN.*

DR. ALBERT A. CINELLI, New York.

"The surest way of preventing pneumonia would be to eliminate the common cold and other mild respiratory infections. In the studies of pneumonia at Bellevue Hospital, 46.7 per cent of the patients gave a history of preceding cold or cough, and careful questioning would probably have shown a much higher percentage." Thus writes Cecil¹ in his textbook of medicine.

We are all familiar with the havoc caused by the common cold and the variety of pathologic complications that follow in its wake. Tubal catarrh, otitis media, laryngitis, bronchitis and sinus suppuration are all common sequelae to coryza. It is therefore quite natural that so many authorities should regard this insidious malady as "man's worst enemy," and to stress the importance of time by therapeutic measures to "nip the cold in the bud."

Without delving into the controversial field of the etiology of coryza, it will suffice to state that a considerable mass of experimental evidence incriminates a filtrable virus which is present in the nasal mucosa during the early stage. It is communicable through droplet infection.

Before giving consideration to the treatment of coryza, I believe a brief review of its symptomatology may be of interest. During the early stage, rhinoscopic examination reveals an edematous, water-logged and inflamed mucosa, with passive hyperemia of the venous capillaries and lymph vessels. The turbinates are often so swollen as almost to occlude the passages, greatly impeding breathing through the nose. The inflammatory reaction is accompanied by migration of leukocytes and transudation of lymph and serous fluid, producing a copious watery secretion. Later a mucus discharge appears, which may finally become mucopurulent.

*From the Nose and Throat Clinic of the New York Post-Graduate Medical School and Hospital.

The three stages of coryza have been divided rather arbitrarily as follows:

The first stage: Period of onset, distinguished by headache, malaise, chills and mild pyrexia, the temperature rarely rising above 102° F.

The second stage: Swelling and congestion of the nasal mucosa with profuse watery discharge.

The third stage: Characterized by mucus, and later mucopurulent, discharge with abatement and complete disappearance of the other symptoms.

Two to three weeks is usually required to complete this cycle. The treatment advocated in this paper is of no value in the advanced stage of coryza and it is therefore essential to bear in mind the different stages of the disease with their individually characteristic objective and subjective symptoms.

Last year my attention was called to a number of clinical reports^{2, 3, 4, 5, 6} extolling the value of homiodin in the treatment of acute and chronic catarrhal infections of the upper respiratory tract. The results obtained by these authors appeared sufficiently interesting and important to warrant verification and a rather exhaustive test of homiodin was arranged for at our nose and throat clinic.

Homiodin is a one per mille aqueous solution of sodium iodide containing traces of free iodine. It is available in one c.c ampoules for subcutaneous administration. The treatment appears to be purely empirical and I have been unable to find a rational explanation as to why this form of iodine therapy should be more effectual than the oral administration of tincture of iodine or intravenous injections of sodium iodide. It is of interest to note that some time ago the sodium iodide content of the solution was reduced from 5 per cent to one-tenth of 1 per cent without in any way influencing its therapeutic value, which therefore must be attributed to the free iodine.

For obvious reasons I was primarily interested in determining the value of homiodin in the treatment of coryza. The cases selected represented all stages, which gave me the opportunity to confirm observations by earlier writers that

the medication is of little or no value when the nasal discharge becomes mucopurulent. The results were particularly gratifying when homiodin was administered during the first stage of the disease. Frequently one injection produced complete symptomatic relief within 24 hours, although I recommend giving a second treatment on the following day. Some patients who did not appear to benefit from the first treatment responded well to the second.

Usually symptomatic relief may be observed very soon after the first treatment. Particularly striking is the subjective improvement, expressed by the patient as a sense of well being, that follows the abatement of sneezing and nasal discharge, disappearance of "stuffiness" and improved breathing. Complete control of subjective symptoms may be anticipated within one to three days after the first treatment. The fact that the resolution may not be completed objectively within this period does not seem to occasion the patient any discomfort, nor have I found it to have any unfavorable bearing upon the prognosis.

In the protracted cases of coryza, the results were likewise encouraging, but here the diagnosis must be conducted with painstaking care before anticipating results with this treatment. If the nasal discharge is at all purulent, homiodin is apt to disappoint completely. Just where to draw the line is at times exceedingly difficult to say, and I feel that only through extensive experience with the medication and infinite care in evaluation of the syndrome is it possible to apply homiodin to the full extent of its usefulness in the treatment of the advanced types of coryza.

Of the many clinicians who have recorded their experience with homiodin, Meyersburg⁶ impresses me as having exercised particular care and mature judgment in his evaluation of homiodin as a means of treating acute and chronic rhinitis. Out of 28 cases of acute rhinitis, uncomplicated and complicated, all but one benefited from the treatment. Twenty-one patients recovered completely within the period of active treatment and six cases were improved. He concludes that "hypodermic injection of free iodine was found effective in conditions where other forms of iodine therapy proved disappointing." His findings correspond with my own observations.

My experience with homiodin in the treatment of vasomotor rhinitis was not nearly as satisfactory as in the acute conditions, and I failed to obtain the uniformly satisfactory results reported by Levine³ and other recent contributors. Straatsma,⁵ on the other hand, reports less than 50 per cent complete cures and while some of the remaining cases appeared to be improved, his observations do not convey the impression that homiodin may always be relied upon to produce gratifying results. I fully agree with him when he states that "it is exceedingly difficult, if not impossible, to predict how a specific case of vasomotor rhinitis will respond to homiodin treatments. The degree of chronicity does not appear to be a reliable guide. In fact, I have observed cases of more than five years' standing respond more readily than have patients suffering from vasomotor rhinitis less than one year."

It is my opinion that the somewhat conflicting results reported with homiodin in vasomotor rhinitis may, in part, be traced to an arbitrary nomenclature. Textbook definitions of vasomotor rhinitis are nebulous and a study of current bibliography reveals a disconcerting variety of interpretations, synonyms and conflicting opinions. But I am here primarily concerned with the treatment of coryza and suffice it to say that homiodin is worthy of consideration as a non-specific measure in the treatment of vasomotor rhinitis.

SUMMARY.

Striking results may be anticipated with homiodin in the treatment of the common cold. The medication is particularly dependable if administered during the first stage of coryza, but it is also of value in protracted cases. Homiodin is likewise indicated as a nonspecific measure in vasomotor rhinitis, but uniformly satisfactory response should not be anticipated. This treatment is of no value in purulent rhinitis.

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1021 Park Avenue.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Boston, Mass., Sept. 16, 1933, during the meeting of the American Academy of Ophthalmology and Otolaryngology. Seventy-nine candidates were examined, of which thirteen were conditioned or failed.

The Board will hold an examination in Cleveland, Ohio, June 11, 1934, during the meeting of the American Medical Association. Also early in the summer (date is not set) an examination will be held in Butte, Mont., in connection with the meeting of the Pacific Coast Oto-Ophthalmological Society. Prospective applicants for certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts building, Omaha, Neb., for proper application blanks.

EYE DISEASES CAUSED BY SINUSITIS.*

DR. NATHAN P. STAUFFER, Philadelphia.

How often does sinus disease affect the eyes? Some patients have blurred vision without opacities or vitreous changes; others complain of intense itching of the conjunctiva; more frequently they complain of headaches without visual or refractive changes, while others are troubled with swelling of the lids, or epiphora. In other cases the ophthalmologist finds retinal changes and desires a report as to the nasal condition. So the rhinologist is frequently asked by the oculist whether a sinus condition is the cause of an eye trouble.

May I present a number of cases illustrating some problems in differentiating or diagnosing sinusitis or eye lesions? All of these patients had X-ray and transillumination and blood examination, with which I shall not burden you.

BLURRED VISION.

Case 1: G. B., white, male, age 33 years, married. This patient had blurred vision with intense itching of the eyes for ten years before he was referred to me by Dr. Isaac Shelly. He had a pansinusitis: He was operated on in the Presbyterian Hospital, where bilateral modified Killian and Caldwell-Luc operations were performed. Diplopia of a high degree developed with a wide divergence of right eye toward his right side.

May 9, 1930, Dr. Langdon reported: O.D., 6/15; O.S., 6/6; superior oblique is involved in the diplopia. Images separated $1\frac{1}{2}$ feet.

July 15: Diplopia now only over right lower field. Separation of images now about three inches.

*Read before Section on Ophthalmology, College of Physicians, Feb. 16, 1933.

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Nov. 1: Fully recovered of blurred vision, itching and diplopia.

EPIPHORA.

Case 2: Mrs. C. T. J., white, age 80 years. Constant discharge of tears, excoriating her cheeks, accompanied by severe headaches. Referred by Dr. Edwin Town and Dr. Webster Fox. This patient had a right frontal, ethmoidal and right maxillary sinusitis with a large growth completely filling her right nares. On April 15, 1931, a modified Killian, ethmoidal and Caldwell-Luc operation was performed in the Presbyterian Hospital and the growth removed. Her sinuses



Fig. 1. Case 2. Epiphora and frontal, and ethmoidal and maxillary sinusitis. Five days after operation.

were filled with pus and the growth was reported carcinoma by Dr. John Eiman. She is alive at the present time and made the quickest recovery of any of my patients. She was discharged in two weeks.

FALSE OBJECTS.

Case 3: W. P., white, male, age 38 years; chauffeur (see Photos 2 and 3). When driving would stop his car for black objects on his right, thinking they were passengers, or for fear of hitting them. Headaches and nasal discharge for years. This patient had a pansinusitis. Under ether in the Presbyterian Hospital I did the usual modified Killian, ethmoidal and maxillary (Caldwell-Luc) operations. He made a

rapid recovery and his black objects disappeared. Three weeks later he noticed he had double vision.

PAIN AND BLURRED VISION.

Case 4: A. E. B., white, male, age 21 years; student at Yale. Unable to study because of severe pains in the eyes and blurring vision. This patient had a left frontal sinusitis and a modified Killian operation, which was performed in the Presbyterian Hospital. After discharge from the hospital he had 14 degrees diplopia, which Dr. de Schweinitz cured by suitable prisms and muscle exercises.



Fig. 2. Case 3. False objects caused by chronic sinusitis; right frontal, ethmoidal, and maxillary sinusitis. Before operation.

This patient required the longest time to cure his diplopia, one year. He is in good health after a period of ten years.

Case 5: Mrs. H., white, age 55 years. Constant blurring and itching. Patient confined to bed with arthritis. Patient has a posterior ethmoiditis with constant postnasal discharge. The question arose, Is her eye condition caused by her arthritic deposits, or her sinusitis? Treatment of the sinusitis relieved the eye condition.

ACUTE CONJUNCTIVITIS.

Case 6: M. B., male, age 8 years; referred by Dr. Heed. History of sudden onset; eyes, conjunctiva, and sclera deeply

injected, profuse lachrimation, which did not clear up under the usual eye treatment so he was referred for rhinological examination. Pus was found under anterior end of both turbinates. Appropriate nasal treatment cured his sinus condition and also his eye troubles.

CHOROIDITIS.

Case 7: T. L., white, male, age 54 years; patient of Dr. de Schweinitz. This patient has a central choroiditis of the left



FIG. 3. Case 3. Taken 15 days after operation on right frontal, right ethmoids and right maxillary sinuses, showing amount of swelling and wound. This shows how little disfigurement there is after a modified Killian operation.

eye. For many years he has had an obstruction of his left nares, due to a deviated septum and a consequent ethmoiditis, maxillary sinusitis and sphenoiditis. He refused an operation.

SWOLLEN EYE LIDS.

Case 8: H. A. L., white, female, age 54 years; referred by Dr. H. Knox. Complained of swelling eyelids and foul breath. Patient had a right frontal modified Killian and right Caldwell-Luc operation in the Presbyterian Hospital. This cured her swollen lids and halitosis.

DROWSINESS AND HEADACHES.

Case 9: P. W., white, male, age 60 years; retired from Pennsylvania Railroad because of headaches and pain in the eye and drowsiness. Patient had polypi blocking both nares, which when removed permitted a free flow of pus from his right frontal and ethmoidal sinuses. I advised him to have a radical frontal operation but he deserted to another physician.

HEADACHES AND DOUBLE VISION.

Case 10: E. S., white, female, age 31 years; referred by Dr. Isaac Shelly. *Complaint:* Unbearable pain over and in the left eye but no swelling, or redness of eye or eyelids.



Fig. 4. Case 10. Pain in eye, severe headaches, unable to work. Radical frontal operation. Cured.

Vision: O.D., 20/20; O.S., 20/70. This patient had a chronic frontal sinusitis, which did not involve the maxillary sinus.

June 6, 1928: In the Montgomery Hospital, with Dr. I. Shelly, I did a modified Killian operation. Patient made a nice recovery and was discharged on June 17. On July 11 she complained of severe pain, which was relieved by a discharge of pus. She wore a bandage over the left eye so long the eyelashes were glued together and the eyelid would not open.

Aug. 8: Was in good health until last night, when she was taken sick with headache and vomiting.

Nov. 9, 1929: An attack of influenza followed by double vision. Pain over the left eye.

Feb. 23, 1930: Pain in left eye and lid swollen shut.

Feb. 24: Left eye completely closed, red, and painful, no pus in nose but X-ray by Dr. Spachman said left frontal was involved.

Feb. 26: In Presbyterian Hospital, under gas and ether, I did a radical Killian on her left frontal. Diplopia, five degrees, treated by Dr. I. Shelly.

Aug. 27, 1931: Headaches cured. No diplopia and has not been to a physician in a year.



Fig. 5. Case 11. Itching of eyes, headache and sinusitis.

ITCHING OF EYES WITH SWELLING ACCOMPANIED BY HEADACHES.

Case 11: Miss S. B., white, female, age 12 years; referred by Dr. E. Town. History: Following a six weeks cold a headache developed with itching and later a swelling of the right upper eyelid, later a large swelling over the forehead. This subsided for several weeks, under boric fomentations, only to reappear. Examination revealed profuse purulent right nasal discharge. Transillumination revealed no right pupillary reflex and dark antrum X-ray by Drs. Spachman and Castner: cloudy right frontal and right maxillary.

April 8, 1931: In Presbyterian Hospital, under ether, a modified Killian and a Caldwell-Luc operation were performed and pus, granulations and polypii were found. The pus per-

forated the external frontal plate and caused the extreme cellulitis of the orbit. She had no muscle imbalance after the operation, although the superior oblique and internal rec-



Fig. 6. Case 12. Suddenly swollen eyelids and sinusitis.



Fig. 7. Case 13. Swollen lid from acute frontal, ethmoidal and maxillary sinusitis.

tus had been removed from their original places of attachment. Evidently the periosteum had been correctly replaced. External appearance similar to W. K. She gained 30 pounds in eight months.

The above cases presented certain well marked premonitory eye symptoms which would have led you to a rhinological examination. In contradistinction to these there are some which have a sudden onset, in which the eye is immediately involved without premonitory symptoms.

Case 12: W. K., male, white, age 9 years; referred by Dr. Cyrus Fridy. Suddenly the left upper lid became swollen; in consultation with Dr. Krall we found he had marked edema of the upper lid but no other eye lesion. Nasal examination revealed a profuse left nasal discharge.



Fig. 8. Case 14. Closed eye caused by acute frontal sinusitis. Anterior view.

He had a modified Killian frontal and Caldwell-Luc maxillary operation with a complete recovery in 1931. The father reports, "He has never had any itching, watery, or blurred or double vision before or since the sinus operation."

Evidently the circulation of the eye was not disturbed until the pus ruptured through into the orbital cavity.

Case 13: A similar case is W. G., white, male, age 27 years, clerk; patient of Dr. Yeager, referred by Dr. Heed. Following a swim in an indoor pool this patient developed a cold; one week later pain over the superior maxilla, which was considered to be neuralgic; the following day his upper eye swelled shut (see Fig. 10). Dr. Heed reported no eye ground changes and concluded he had a sinusitis. Nasal examination revealed a purulent nasal discharge, right side; X-ray and transillumi-

nation confirmed our diagnosis of a frontal and ethmoidal and maxillary sinusitis. A modified Killian frontal and Caldwell-Luc maxillary operation cured his eye condition and sinusitis. He, however, developed a diplopia, for which he was referred to Dr. Heed, who cured him with appropriate treatment, which he will describe.

Case 14: J. K., white, male, age 8 years; referred by Dr. C. Vetkoskey. Following an acute infectious disease suddenly developed a swelling of the left upper eyelid. Conjunctiva red and swollen, out eye grounds negative. Nasal examina-



Fig. 9. Case 14. After operation for left frontal and left maxillary sinus operation.

tion revealed a purulent left nasal discharge. X-ray and transillumination confirmed the diagnosis of a frontal and maxillary sinusitis. Under general anesthesia in the Presbyterian Hospital I did a modified Killian frontal and a Caldwell-Luc maxillary operation. He made a complete recovery.

The last three cases illustrate how a concealed abscess can go for days without affecting the eyes until perforation of a plate will permit the pus to escape into the orbital cavity, their chief symptom being pain referred to either the frontal region, or a general headache. The patient, or family, has usually considered these latter cases as eye conditions for which they were sent to the oculist, who recognized the sinus condition and referred them to the rhinologist. Needless to

add that every oculist confronted with an eye complaint, which upon examination does not show any well defined reason within the eye, immediately suspects the presence of a nasal disease.

Fifty years ago these people might, and probably would, have lost their sight, or their lives.

These patients demonstrate very clearly how sinusitis can cause various lesions of the eyes. Some of these patients were only annoyed by their eye symptoms, while others were rendered miserable, or totally incapacitated by their disease. The question naturally arises, how did the sinus disease affect the eye—by disturbing the blood supply, that is by pressure, or by continuity or by blood stream infection, or by lymph channels; or a systemic toxemia.

Case 1: Evidently had his itching and blurring from a toxemia.

Case 2: Surely had her epiphora from blockage of lacrimal duct.

Case 3: Probably his false objects came from a toxic condition.

Case 4: Blurred vision and eye pains due to toxemia and pressure.

Case 5: Blurred vision and itching caused by toxemia.

Case 6: Inflamed conjunctiva and sclera and lacrimation due to blood stream infection.

Case 7: Choroiditis probably caused through blood stream or lymph channels.

Case 8: Swollen lids possibly caused by continuity (posterior ethmoiditis).

Case 9: Headaches, drowsiness and pain in eyes caused by obstructed drainage and septic absorption.

Case 10: Severe eye pains and headaches caused by pressure.

Case 11: Itching of lids and edema produced by disturbed blood supply and infection.

The patient will want to know: 1. His chance of cure; 2. his visual acuity; 3. his personal appearance.

1. As to sinus disease, he can be cured if osteomyelitis is not present and if he has patience to permit the necessary operations, however many that may be.

2. His vision should be unimpaired, although he may have a temporary diplopia if the frontal operation is done.

3. His personal appearance should be the same as before the operation if the proper precautions are observed.

1900 Rittenhouse Square.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Following is a list of the officers of the Los Angeles Society of Ophthalmology and Otolaryngology for 1934:

Dr. F. H. Brandt, President.

Dr. Walter R. Crane, Vice-President.

Dr. Pierre Viole, Secretary-Treasurer.

Dr. Julian Dow, Committeeman.

Dr. Dean E. Godwin, Councilor.

The meeting place is the permanent quarters of the Los Angeles County Medical Association, 1925 Wilshire boulevard, Los Angeles. The meeting dates are the last Monday of each month.

DETECTION OF SIMULATED DEAFNESS.

DR. ROLAND D. RUSSELL, Chicago.

How frequently an otologist sees a case of simulated deafness varies considerably according to the source of his practice. If his work is referred chiefly by family physicians, he will not see many cases, and these may be for the most part hysterical patients, rather than malingerers. In a practice made up largely of cases into which the question of compensation enters, the number of cases of simulated deafness must be relatively large. In a practice of this nature there will be more cases of malingerers than of hysteria.

In cases of apparent deafness several conditions must be differentiated. Hysteria and malingering, though detected by similar tests, are not to be confused. The hysterical patient actually believes that he is deaf, but the malingerer knows that he is not deaf, but deliberately attempts to deceive. The hysterical person tries to excite sympathy, and usually presents other symptoms, such as corneal and pharyngeal anesthesia. The stupid and mentally clouded person must be differentiated from the deaf patient, for often the stupid person appears deaf, and as often the deaf person gives the impression of being stupid. I was recently called by one of my associates to observe a negro patient who repeated words after the examiner with an interval of about ten seconds. Had the examiner been less patient or less alert, the case would have been called one of deafness.

There may be no actual defect in the hearing acuity, or there may be an exaggeration of an actual defect. Because of the hardship imposed on one in simulated bilateral deafness, it is less frequently encountered than the unilateral type. This is fortunate for the otologist, because bilateral simulation is more difficult to detect.

Every case of apparent deafness where compensation of any nature is involved is to be looked upon with suspicion,

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and the examiners' actions governed accordingly. The examiner must not show by his manner that he suspects the patient of malingering, but on the contrary must appear quite sympathetic in order to throw the patient off his guard. Once the examination is finished the examiner must not tell the patient why he knows that he is a malingerer, for each time an intelligent malingerer is examined he learns something about the tests, and the examiner must not aid the patient to avoid the pitfalls in the future.

Due partly to the fact that malingerers make a study of hearing tests, either from books, from actual experience in being examined, or from being tutored, and partly to the fact that a single test does not always prove that the case is or is not one of malingering, a number of methods have been devised.

The patients' reactions must be carefully observed. The malingerer makes exaggerated attempts to hear, while the truly deaf individual makes no great effort to hear. In voice tests the malingerer repeats words which have no similarity in sound to those spoken by the examiner, while the truly deafened person responds with words of similar sound to those spoken by the examiner. The malingerer is frequently antagonistic to the examiner, but the truly deafened person is very rarely so.

It is said that persons with bilateral deafness usually respond to tactile impressions so that on dropping a tin basin or similar object behind a really deaf person he will look around, whereas a schooled malingerer will take no notice. This is not a conclusive test and should be backed by other evidence.

The individual complaining of pronounced bilateral deafness may be detected often by calling him from the reception room in an ordinary voice. If he responds readily, but on conducting the voice tests in the examining room he can hear at say only one or two feet, obviously he is attempting to deceive.

If on conducting the voice test the subject shows a marked impairment of hearing, but at a distance of say thirty or forty feet he obeys readily the command to rub or plug the opposite ear or to cease rubbing or plugging the ear, he is

exposed. This test is applicable to either bilateral or unilateral simulated deafness.

A useful test in severe bilateral deafness is a modification of the Lombard test. A Barany noise apparatus is sounded in both ears, simultaneously while the patient is reading. If the patient elevates the voice, he is not sincere.

Another good test in cases of simulated bilateral deafness is to have two or preferably three or more examiners in the same room. The patient is engaged in a friendly conversation by an examiner at close range, and after the conversation has gotten well under way, the other examiners, who are several feet distant from the patient, comment or ask questions. If the patient responds or begins the response and suddenly stops, he is unmasked. In a similar manner, in tests conducted by a single examiner, a sudden question asked at a distance greater than that at which the patient admits hearing, and while the examiner appears to be engaging his attention in something besides the patient, will often be answered.

In cases of both partial bilateral and partial unilateral deafness, the same words may be used a number of times, and the variations noted. If there are marked discrepancies, the patient is probably attempting to deceive. However, it should be noted that after a deafened patient is thoroughly familiar with the words being used, he repeats them more readily. In a similar way a number of audiograms may be taken and compared. In taking audiograms it is well not to begin either at the uppermost or lowest note, and descend or ascend octave by octave, but rather skip about, taking a high, then a low note, so that the patient is less able to simulate a graph typical of any type of deafness.

Disparaging remarks may be made in the patient's presence, watching the patient's facial expression and the presence or absence of flushing.

A type of case sometimes coming under observation is one in which the subject claims to be a lip-reader. One may detect this case by asking questions without facial or lip expressions; that is, with the lips immobile and opening and closing the mouth the least possible. From time to time, while asking questions, one may pretend to rub the nose in order

to hide the lips. Finally the patient is taken into the dark room under pretense of transilluminating the sinuses, and while doing this a casual conversation is carried on and the patient's response noted.

In unilateral deafness the ear speculae test is carried out with four speculae, two plugged with wax and two unplugged. Atomizer tips are better, for they may be pushed tightly into the ears without danger of excoriating the external auditory meati. This test is begun with two open tubes and words repeated at varying distances and the results noted. The tests are repeated with one open and one closed tube, again with two open tubes, and then with one open and one closed tube, and the results noted. If the patient refuses to hear the voice at a distance at which he has previously heard it while testing the ear exposed, he is not frank in his responses. In this test care must be exercised not to stigmatize the mentally dull patient as a malingerer. I recall a case in which the voice tests had indicated that the patient was totally deaf in one ear, and that the hearing was normal in the other ear. On plugging the good ear with a hollow nasal tip the patient would not repeat words. At the moment the patient was thought to be a malingerer, but in examining the eyes, both optic discs were found to be choked, and caloric tests showed a complete suspension of the vestibular function. The case was one of brain tumor.

Another test used by Weber is to read a story to the patient who has his eyes bandaged. The story is made up of words in capital letters and words in small type. The words in capitals are whispered and the words in small type spoken. The story is read at a distance at which the patient hears the spoken voice without trouble. The malingerer becomes confused and either repeats the whispered words or does not repeat the spoken words.

The stethoscope test is performed with a stethoscope, one earpiece of which has been plugged with wax. The plugged ear piece is placed in the normal ear and the open one in the supposedly deaf ear, and words spoken into the stethoscope. The stethoscope is now removed and the "deaf" ear tested with the voice. If the patient heard the voice spoken into the stethoscope and fails to hear it in the deaf ear without the stethoscope, he is probably malingering. One must be

certain in this test that the good ear was tightly plugged and that the voice was not too loud. To check this one should close the bell of the stethoscope and repeat the test. If the patient hears now, he was not malingering.

If a person has good hearing in one ear and is totally deaf in the other, he is still able to hear a loud voice at close range with the normal ear tightly plugged. If he does not, he is not frank in his responses.

Callahan's test is performed with a stethoscope with two tubes of unequal length. The earpiece connected with the shorter tube is placed in the deaf ear, and the earpiece connected with the longer tube is placed in the good ear. If the hearing is good in the supposedly affected ear, the sound in the opposite ear will be masked, and the subject will have the impression of hearing the voice in the deaf ear. If the patient is malingering, he will state that he does not hear the voice.

One of Becker's tests is performed also with a stethoscope with two tubes of unequal length. The sound will be lateralized to the ear connected to the shorter tube, and the ear connected to the longer tube will be masked completely so that the patient has the impression that the sound is monaural when it is actually binaural. Compressing the longer tube will produce a decided change in the sound because binaural hearing is then converted into monaural. The shorter tube is connected with the good ear and the longer tube with the affected ear. A loud ticking watch is placed at the bell of the instrument. Then the tube to the affected ear is compressed. If this produces a change in the sound in the good ear, the presence of binaural hearing is established.

Another test devised by Becker depends on the principle of lateralization of sound to the ear at which the sound arrives with greater intensity. For this test a piece of rubber tubing four feet long with olive tipped ear pieces inserted in each end and a loud ticking watch are needed. If the ear tips are inserted, one in each ear, and a loud ticking watch pressed against the middle of the tube, the sound will be heard equally in both ears. If the watch is moved a short distance away, it will be lateralized to the nearer ear. The "normal" ear is first tested. One end of the tube is inserted into the "normal" ear. The other end one or two inches from

the olive tip is pressed with the watch sufficiently to almost compress the tube, and the patient is asked if he hears the ticking. If he hears it distinctly, one proceeds with the test; if he does not, the watch is moved up the tube one or two inches at a time until it is heard, and the point at which the watch is heard is marked. Both tips are now inserted, and the tube dropped back of the patient's head. The examiner stands back of the patient, and applies the watch to any point between the middle and the supposedly deaf ear, provided this point does not fall short of the place marked in the preliminary test for the sound ear. The deafened person will state frankly that he hears the sound in the good ear. In the case of the malingerer, the sound is lateralized to the "deaf" ear so that he is at a loss to answer, and states that the sound is not heard or hesitates long before stating that he hears it in the sound ear. Becker states that the tube toward the sound ear may be occluded by a screw clamp, and, if he still hears the instrument in the sound ear, the patient is exposed. The tube toward the good ear may be occluded by a clamp at the beginning of the test; if the sound is not heard, and on gradually releasing the clamp is still not heard, he is again exposed.

In cases in which the patient states that he is deaf, say in the right ear, refers the Weber test to the left ear, and on placing the fork on the right mastoid refers the sound to the left, Becker directs a stream of air against the left drum membrane, rendering the left cochlea functionless during the time that the air is being used. The malingerer will state that he hears the forks in the left ear on placing it on the left mastoid. On placing it on the right mastoid he either does not admit hearing it or refers the sound to the left.

Erhard's test is performed by placing the patient in the middle of a large room. The ear supposed to be deaf is plugged and the watch gradually brought toward the normal ear, and the patient asked to count the beats. The normal ear is then closed and the test repeated. Normally a loud ticking watch can be heard at a distance of three or four feet; therefore, if the patient states that he does not hear the watch at two or three feet with the normal ear closed, then he is attempting to deceive.

The coin click test is performed by testing the distance at which the patient admits hearing the coin click with the eyes

open. The eyes are then bandaged. If the patient hears the click of say 10 feet with the eyes open, examiners are stationed at 10, 25 and 40 feet. The patient is instructed to count the clicks and all the examiners are to click their coins as many times as the first examiner. If the patient hears twice or three times as many clicks as the first examiner made, he is unmasked. If the patient counts only the first examiner's clicks, the second or third examiner is signalled to click his coins alone.

Lombard's test is performed as follows: The patient is told to read aloud, and after noting the loudness of the voice, a Barany noise apparatus is sounded in the "good ear." If the patient is really deaf in the opposite ear, masking his good ear will cause him to elevate the voice markedly. If the hearing in the unmasked ear is good he is able to estimate the loudness of his voice, and therefore will continue to read in approximately the same tone of voice.

In unilateral deafness the Barany apparatus may be sounded first in one ear and then in the other as the examiner talks to the patient. Then the examiner places it in the "deaf" ear, saying: "You don't hear that, do you?" and then in the "good" ear, saying: "But you hear it now." If the patient answers a question while the noise apparatus is being sounded in the "good" ear, he is attempting to deceive.

Teal's test: If in performing the usual tuning fork test, the patient denies hearing the forks by air conduction in the "deaf" ear, he is blindfolded and the bone conduction over the mastoid on the "deaf" side is tested. Usually perception by bone conduction is admitted; if not, the correctness of his responses is open to question, as with one good ear the sound should be conducted through the skull to the opposite ear. He is told that the test is to be repeated. A non-vibrating fork is placed against the mastoid of the deaf ear and a vibrating fork of the same pitch as that previously used is held a short distance from the affected ear. If he admits hearing the fork he is discovered. I have not found this test very satisfactory, as most persons are able to distinguish between bone and air conduction.

Another fork test used by Weber is performed as follows: Pinch 256 d. v. fork and wait say twenty seconds, then strike

another fork so that it clicks noticeably and discard it. Place the fork which has been vibrating for several seconds at the meatus and note length of time that it is heard. Repeat the same procedure with bone conduction. Then repeat, allowing patient to hear the fork from the first and note discrepancies. If the patient hears the fork in both instances the same length of time, he is malingering.

The Stenger test: When two sounds of the same pitch and intensity are presented to the ears, and the source of the sound in one ear is nearer than that of the sound in the opposite ear, the subject will be conscious of hearing in the ear toward the nearer source of sound. The test is performed with two forks of the same pitch, and the patient is blindfolded. First a determination of the distance at which the patient can hear the fork distinctly in the good ear is made. If he denies hearing the same fork in the "deaf" ear, and hears it at say ten inches in the good ear, one of the vibrating forks is held at say three inches from the "deaf" ear and another fork is brought up to say six inches of the good ear. If the patient now says that he hears no sound at all, even though he previously heard the fork at ten inches, he is obviously malingering.

The Wells test is performed with a piece of rubber tubing, one-fourth inch in calibre, thirty inches in length and of sufficient firmness to conduct sound well, into one end of which the stem of a tuning fork has been inserted tightly and into the other end an ear piece which fits firmly into the external auditory meatus. The fork in the rubber tube is vibrated and the other end of the tube inserted into the "deaf" ear. Another fork of exactly the same pitch is brought toward the good ear to a point at which the patient has previously heard the fork distinctly. The really deaf person will state that he hears the fork as soon as it comes within the range of hearing of the good ear. The malingerer will not admit hearing the fork at all.

The Chimani-Moos test is performed as follows: A fork suitable for testing bone conduction is held alternately at an equal distance from each ear, and the patient is asked in which ear he hears the fork more distinctly. He will indicate the good ear. The fork is now placed on the skull in the midline and the patient again asked in which ear he hears the

sound. The malingerer will state that he hears it in the good ear, while the individual with deafness due to middle ear disease will state that he hears the sound in the deafened ear. The patient is now told to plug the good ear with the finger. The malingerer will probably say that he no longer hears the sound, or hears it faintly. In my opinion this is one of the tests of the least value, and in the majority of cases in which it is positive, it only demonstrates the unreliability of the Weber test in the particular case under consideration; however, if backed by other evidence of malingering it forms a link in the chain of evidence. I say that the test is not conclusive because I have seen many cases of undoubted deafness give positive responses of malingering to the Chimani-Moos test.

The cochleo-palpebral test of Gault is performed by occluding the good ear and then making an unexpected noise near the "deaf" ear. If there is a slight contraction of the lid, hearing is indicated. This test is also of value in bilateral "deafness."

If in performing the caloric test, normal vestibular responses are obtained, the presence or absence of deafness is not proved, for in conduction types of deafness one expects to find normal vestibular reactions, and in nerve deafness the cochlear division of the eighth nerve is frequently affected without any involvement of the vestibular division; on the other hand, if vestibular reactions are absent or markedly depressed, much credence is to be placed in the patient's statement that he is deaf. The caloric test has some value as a means of suspending the cochlear function of one ear while testing the hearing in the other ear in cases of absolute or nearly absolute unilateral deafness. If while irrigating the normal ear the patient answers questions, as, for example, as to the presence or absence of vertigo, it is shown that he is not really deaf in the supposedly affected ear.

Again allow me to repeat that each time an intelligent malingerer is examined, he learns something about the tests, and to warn the otologist against instructing these malingerers, by letting them know by either word or manner at which point they have been detected. On the contrary, a few more tests should be done after detection, in order that the malingerer shall not suspect just what response trapped him.

The examiner must be careful not to stigmatize a patient as a malingerer on mere suspicion. Then, too, the fact that the examiner is convinced that a patient is malingering is not sufficient, for many of these cases are potential court cases, and the otologist must be prepared to defend his opinion on the witness stand.

55 East Washington Street.

A TEST FOR SIMULATION OF DEAFNESS.

DR. CHAS. FIRESTONE, Seattle, Wash.

It has long been known that the detection of either unilateral or bilateral simulation of deafness has been relative at best; rarely is it absolute. Even in our dealings with patients who have no cause to be suspected of simulation, we find these patients registering the perception of the lowest frequency tuning forks, positive Rinne and Schwabach, yet the patient himself is conscious of a deficiency in hearing not demonstrable objectively. We must deduce in cases like these, and we all have them, that these patients are not malingerers, but that armamentoria have not been developed sufficiently to objectively register their complaint. By our standards we designate such patients as having unimpaired hearing, and frequently label them as suffering from a neurosis.

In the detection of bilateral simulation of deafness our technique is largely circumstantial in the early stages. A claimant who states that he does not perceive the vibrations of a C4 tuning fork on either ear presents indeed a problem very difficult of solution in its early stage. Here we have to depend largely on gleanings from detections on the claimant while he is unaware of such detections. The labyrinthian reaction to various stimulations will be of little help early after an accident. Its normal response will not condemn the claimant, as deterioration may take place and become complete as late as 2 years following the injury. Failure of response, conversely, is no corroborative; as an exudate in the labyrinthian capsule may be later absorbed, and the reaction returned to normal following the absorption. The author has seen cases illustrating both these statements. The subsequent life or death of the labyrinth is largely the key to the truth. But as stated above, early, we can not with certainty evaluate the absolute state of the claimant's condition.

When we come to the detection of unilateral simulation of deafness we reach easier ground. Every recognized func-

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tional test leaps to our aid and a relatively more accurate estimation may be made, although in the opinion of this writer this estimation can never be conscientiously interpreted as absolute. This author has used the several tests classically employed for such purposes. The Stenger tests have been found most useful, and the others have proven mostly corroborative in the hands of the author.

The author has devised and for the past three years has been using a test which combines bone conduction and air conduction, both applied simultaneously to the allegedly impaired ear, while in synchrony with this, the unimpaired ear is tested for air conduction alone, by means of a synchronously vibrating tuning fork. The rationale underlying this procedure is simple, and its application may be carried out by one versed in these tests without costly armamentoria. It can easily be verified by applying the test to oneself.

The rationale underlying this procedure is that, in combining bone conduction with air conduction and applying it simultaneously to the allegedly impaired ear, the examiner sets up an intensity of vibrations, which, if perceived by an allegedly injured ear, are perceived with such intensity that they render the examinee unable to localize vibrations perceived in the good ear from the synchronous tuning fork, reaching it simultaneously by air conduction only. It brings into the field a positive finding to go on and that is that the perception in an allegedly injured ear of the vibrations via the twin conducting source produces a positive inability of the examinee to localize synchronized vibrations reaching the good ear from an air conduction source only. This is based on established principles of functional testing long in vogue. This procedure segregates the malingerer from the non-malingerer at a glance, and as one progresses with the testing, it serves as a definite indicator of the amount of injury sustained by the examinee, if any.

Technique: The technique of this test is a simple one, and can be carried out by any aurist. The first and one of the essential portions of the test is to do and record a routine functional test of both the injured and uninjured ear. The record of the functional ability of the good or uninjured ear is even more important than the record of function of the injured ear, inasmuch as the examinee will be anxious to

demonstrate his inability to function with the injured ear, and will be more than anxious to prove that his good ear has keen perception in an endeavor to impress the examiner with the comparative hardness of hearing in his injured ear. It is on the hearing in the good ear that the ultimate evaluation of loss of hearing in the injured ear will rest.

Apparatus Required: Synchronous tuning forks C-64, C-128, C1-256, C2-512, C3-1024 and A-440, and an occlusion bandage for both eyes. Tuning forks with spherical handle tips as shown in the accompanying sketch are preferable. The author has been able to obtain some of the forks with such spherical tips.



Tuning Fork with Sphenoid End.

The examinee is seated on a stool and the occlusion bandage is applied in a way to obtain absolute occlusion of both eyes. The object of this is to keep from the examinee the knowledge of his good ear being tested. In the Stenger test we strive to keep from the examinee the knowledge that his impaired ear is being tested. The examiner stands behind the examinee. The examinee is told to raise his right index finger when he hears the vibrations of the tuning fork after he has been apprised of what he is expected to hear and keep it raised as long as he continues to hear it.

The test is usually begun with the C-64 synchronous tuning forks. Both tuning forks are set vibrating with equal intensity and are allowed to vibrate for a very brief while in order to minimize overtones. The end of the handle of one tuning fork is then gently pressed to the posterior wall of the external canal of the allegedly deaf ear close to the external auditory meatus. With the vibrating tuning fork in this position, test the good ear for perception by air conduction of the other tuning fork which is vibrating in synchrony. The allegedly impaired ear will be receiving sound waves from the twin source of air conduction in combination with

bone conduction, one to the intensification of the other. If the allegedly impaired ear perceives the vibrations by both the bone and the air conduction, they will be perceived so accentuated that they will render him unaware of the weaker vibrations reaching his good ear from an air conduction source only. The examinee, being aware only of his injured ear being tested, if he is malingering, will naturally deny the perception of any of the vibrations. If he perceives only the vibrations from the bone conduction and not those from the air conduction in the allegedly impaired ear, or if he does not perceive either one in the allegedly injured ear, he will immediately become aware of his good ear being tested and signalize the perception of the vibrations, localizing them to his good ear.

If he is found to be malingering at first, continue with the vibrating forks in place until bone conduction has ceased or has become so weak as to be of little or no intensification of waves emanating from the same tuning fork and being transmitted by air. We will thus establish a situation wherein synchronous waves are reaching both ears by air conduction only. If he does not signalize his perception of them, and his good ear is known from earlier functional test to perceive the vibrations of this particular tuning fork, it may be logically deduced that he is perceiving these vibrations in both ears, but is unable to localize them. Thus we have a distinct method of finding out whether the examinee perceives by either bone or air conduction.

If he is found to be malingering we can establish the fact absolutely and immediately by keeping the tuning forks in position and terminating the vibrations of the one pressed against the posterior wall of the external canal by pressing on the prongs. The examinee will immediately become aware of the vibrations reaching his good ear by air conduction and localize them, vibrations which he has been perceiving all the time, but has been unable to segregate and localize because of the intense vibrations reaching his allegedly impaired ear taking the foreground.

The examinee is tested beginning with the lowest set of forks, progressing up to the highest, until the tuning fork is reached which he hears in the injured ear. How will we know when we have reached this particular tuning fork?

We will know it by the fact that the examinee will for the first time be unable to localize the vibrations reaching his good ear by air conduction only from its synchronizing mate.

It was stated earlier in this discussion that the gross malingerer may be segregated almost immediately by using this procedure. How is this arrived at? If he is unable to localize the C64 tuning fork to his good ear while this procedure is carried out, and the C64 tuning fork, as stated earlier, is the first one tested for, we immediately conclude that his allegedly injured ear is perceiving these vibrations and of course the examinee has denied hearing them during the functional test routinely performed prior to this procedure.

What of the claimant who as a result of an injury was left with a small amount of middle ear deafness and as a result of this condition contends that he is totally deaf in the injured ear? He vindicates his contention by denying the perception of any of the tuning forks with which he is routinely tested, but admits rather boastfully that he perceives every one of the tuning forks with his uninjured ear, going even further and stating that prior to injury he could hear as well with his now allegedly deaf ear as he can now with his good ear.

Here again this test has proven of value in the hands of the author, for when we remember that air conduction lasts longer than bone conduction (Rinne), we can apply this test and get a logical deduction from our findings. In a condition of this sort, when bone and air conduction are simultaneously applied to the injured ear, while only vibrations by air conduction from a synchronously vibrating tuning fork reach the good ear, the latter will be perceived rather faintly at first, become pronounced as the vibrations reaching the impaired ear by bone conduction diminish in intensity and will be heard clearly and distinctly when the bone conduction to the impaired ear has ceased. We would naturally expect this inasmuch as air conduction lasts longer than bone conduction. In the exemplary case cited above we can actually find the amount of impairment of hearing a claimant has sustained, regardless of his keenness or of the amount of coaching he may have received prior to examination. If he responds as stated above to a given synchrony of tuning forks,

he is tested with synchronous tuning forks of the next higher frequency and so on up until the fork is reached that he perceives in his injured ear by both bone and air conduction. How do we know when we have reached this tuning fork? From his inability to localize in the good ear by air conduction the vibrations of the tuning fork vibrating in synchrony with the tuning fork applied to the injured ear and transmitting its vibrations by both air and bone. He will either deny hearing anything or give a confusion of answers, and we know from previous examination of his good ear that he does perceive these vibrations.

What of a claimant who is totally deaf as a result of an injury? As stated earlier, the labyrinthian reactions may and may not be of absolute help in these cases. It is therefore not totally reliable. Just as in the case where the claimant totally malingered so in this case, the truth may be reached in very short order. The vibrations reaching this deaf ear from both bone and air conduction are not perceived by this ear. These vibrations are perceived by the good ear and intensify the vibrations already reaching this ear via air conduction from the tuning fork vibrating in synchrony. The examinee will immediately localize to the good ear and will notify the examiner that his perception in the good ear is accentuated. In other words, he will say he hears clearly with his good ear.

Here then we have enumerated three types of conditions of unilateral deafness or pseudo-deafness; the total malingerer, the partial malingerer and the claimant who has actually been rendered deaf unilaterally as a result of an injury. These comprise all the cases of unilateral impaired hearing that are referred to us for rating. It will be seen from these types that this test serves directly a two-fold purpose. It absolutely segregates the malingerer from the non-malingerer and gives us a direct yardstick to measure the amount of injury actually sustained.

Advantages of this test: From a first analysis of this paper one may deduce this procedure to be a revamping of the Stenger method. A little further study, however, will reveal several features in this test not possessed by the Stenger or any of the other tests known to the author.

1. Many malingerers develop a very keen threshold of localization and delude our expectation from the Stenger test. In other words, they are alert to the removal of the vibrating tuning fork from the good ear, and becoming aware of such removal, state that they do not hear the vibrations any more, while of course they are hearing the vibrations in the allegedly deaf ear. In this method, if the injured ear hears a given tuning fork, it hears it from the twin source of bone and air conduction, and renders the good ear unable to detect the vibrations of the synchronous tuning fork reaching it by air conduction alone. In this method the examinee is not even aware of his good ear being tested if he is malingering. In other words, it establishes the existence of hearing of a given tuning fork in the injured ear by the fact that the examinee is unable to hear simultaneously with his good ear the synchronously vibrating tuning fork reaching it by air conduction. It is positive.

2. In cases of unilateral nerve deafness in which the labyrinthian reaction is not completely corroborative, the bone conduction originating from the vibrating tuning fork applied to the injured ear will serve to strengthen the air conduction from the synchronous tuning fork applied to the good ear and an intimate acknowledgment of the perception in the good ear will be obtained.

3. In cases of injury impairing the middle ear, bone conduction will be increased to that ear. In no case, however, is bone conduction as clearly perceived as is air conduction when it is perceived, nor does bone conduction last as long as does air conduction. The examinee will therefore acknowledge the perception of the air conduction in the good ear until we reach a tuning fork which both ears perceive by air conduction. He will then be unable to localize it. It also affords us a measure for the amount of shortening of air conduction of a given frequency of vibrations if such shortening exists.

4. Since we can test with a wide range of tuning forks, a more accurate determination may be made than with the Stenger test, which uses only the A-440 synchrony of tuning forks.

5. To a certain extent it provides us with a means for direct computation of the percentage loss of hearing in a given case.

6. It affords a direct means of comparing the duration of air conduction perception in the injured ear to that of the good ear.

Conclusion: The above described test, in the opinion of the author, represents a valuable adjunct to the tests employed at present to apprehend the individual simulating deafness. It adopts for its rationale several well founded principles in functional testing which have not been applied to malingering tests. In the author's experience with industrial malingerers in the past three years, he has found this test of primary importance in arriving at an evaluation of the state of hearing following a given injury.

537 Stimson Bldg.

UNILATERAL LOSS OF HEARING FOR HIGH SOUNDS
FOLLOWING HEAD INJURY. INTERNAL EAR
DEAFNESS. PROBABLE FRACTURE OF LEFT
TEMPORAL BONE. TEMPORARY PARALY-
SIS OF FACIAL NERVE. CASE
PRESENTATION.*

DR. PHILIP S. STOUT, Philadelphia.

Private patient, Ella R., age 30 years, white, American; widow, two children, ages 10 and 11 years. Father and mother living and well. Two brothers and two sisters living and well. No deafness in family. Had measles and appendicitis. Never had discharging ears. As far as she knows never had any deafness in either ear before the accident. Did have some trouble with left eye from childhood. Referred by Dr. LaBarre Jayne Leamy.

History of Accident: On Jan. 23, 1933, while riding with three others, the car was struck by a truck and badly wrecked. She was knocked unconscious and was taken about eleven miles to the Pottsville Hospital. Man sitting on seat next to her was killed. She did not regain consciousness until she arrived at the hospital, which, I should judge, was nearly an hour. Her first impression was of very severe pain over her forehead and pain over her left ear with blood flowing freely from the left ear. Later she vomited black blood. No bleeding from the nose or mouth. From time to time she would have sharp, shooting pain in the left ear.

Three days later she noticed a numbness and peculiar pricking sensation of the external canthus of the left eye and the left side of the upper lip, then complete paralysis of the left side of the face and inability to close the left eye. The paralysis lasted three and one-half weeks, when it improved and is now, seventy-two days after the accident, practically well. There was also loss of taste and numbness of the left side of the tongue. This has also improved.

*Read before the Philadelphia Laryngological Society, April 3, 1933.

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Examination of ears, March 7, 1933, forty-one days after the accident: Right ear: External canal negative. Drumhead negative. Audiometer readings, normal hearing. Left ear: Clotted blood in external canal. External canal swollen in posterior part and fresh blood exudes from what appears to be a tear over the swollen area. The drumhead is partly covered with clotted blood (black) and through the drumhead appears to be something dark either in the drumhead or the middle ear, probably blood. Audiometer readings show complete loss of 8192 and about 30 per cent loss of 4096 (from 64 to 2048 is normal). This was the same on subsequent tests. Bone conduction showed it to be internal ear deafness. Several subsequent audiometer tests gave the same readings. Vestibular: She had and now has a slight giddiness. Occasionally she had to take an extra step to the left to keep from falling. No Barany was done nor did we feel that a caloric was justified under the circumstances. No spontaneous nystagmus found.

Transillumination: Sinuses negative. Right mastoid clear, left mastoid dark. Tonsils diseased. Nostrils free, no blood or evidence of disease. Eyes tested by Dr. E. B. Miller. Summary: Vision, O. D., 5/5; O. S., 5/60.

There is no evidence of any gross lesion in the left eye due to the automobile accident. Had poor vision in the left eye from childhood.

X-ray made right after the accident did not show a fracture of the skull.

Examination, April 8, 1933, seventy-five days after accident, there was still some oozing from the posterior wall of the external canal.

Summary: Young woman had a severe accident, struck over left side of head, unconscious for about one hour, had severe frontal and left side head pains, bled freely from left ear for two days, some blood still exuding several months later, with partial obstruction of the external canal by a drooping of the posterior part of the canal, paralysis of the VIIIth nerve and the chorda tympani nerve on the left side, with recovery after three and one-half weeks, persistent loss of the high sounds on the same side, both air and bone conduction, the opposite side being unaffected.

Presentation of patient.

269 S. 19th Street.

RECURRENT MASTOIDITIS WITH PETROSITIS,
TEMPOROSPHEOIDAL ABSCESS, LARGE
EPIDURAL ABSCESS AND RECOVERY.

REPORT OF A CASE.

DR. CLARENCE H. SMITH, New York.

The following is the report of a case of recurrent mastoiditis with petrositis, temporosphenoidal abscess, large epidural abscess and recovery. Bernard B., age 12 years. Right simple mastoid after scarlet fever, six years ago. Complete healing in six weeks.

March 10, 1933: Mastoid cavity broke down and abscessed after acute purulent otitis. Incision and drainage.

March 25: Brought to hospital with these symptoms: Violent headache, vomiting, restlessness, temperature 103° , drowsiness. There was considerable neck rigidity, positive Kernig. Cerebrospinal fluid under increased pressure, cloudy, 8,400 cells per 1 cm.m., no organisms.

Diagnosis: Protective meningitis. The mastoid wound was revised, dura and sinus extensively exposed.

March 27: The general condition was as before, symptoms somewhat relieved by lumbar puncture. Cell count, 3,200.

March 29: Symptoms persistent and relieved only by lumbar or cisternal puncture.

April 1: Complained of pain around right eye, paroxysmal and severe.

April 4: Pain around right eye persisting and condition not improving. Wound was reopened; radical mastoid operation. Tegmen antri and tympani removed. Blunt instrument inserted about $1\frac{1}{2}$ inches along roof of petrous pyramid, in direction of apex, searching for epidural abscess; none obtained.

April 6: Headache, vomiting, yawning, drowsy, dilated right pupil, weakness upper and lower extremities left, left

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lower facial weakness, left abdominal reflexes diminished, left ankle clonus, left Babinski. No hemianopsia; two diopeters papillitis.

Diagnosis: Right temporosphenoidal abscess.

Operation: Right temporosphenoidal lobe aspirated through mastoid wound, from below upward. About two-thirds ounce pus obtained. Rubber catheter inserted and sewed in wound.

April 12: Catheter replaced by Mosher drain.

April 15: Since last operation the right frontal pain has been increasing in severity and in the number of paroxysms. Patient apparently extremely ill, lying in semicomatose condition, waking up occasionally to shriek with pain around his right eye.

Operation: Instrument again inserted along roof of pyramid, hugging the bone and, of course, beneath the dura. At a depth of about $1\frac{1}{2}$ inches an abscess was entered and pus in large quantity, over two ounces, escaped. A rubber tube was inserted to drain this abscess.

April 27: Mosher drain removed and replaced by small rubber catheter.

May 3: Drain removed from brain; no further discharge.

May 6: The drain to epidural abscess has been pulled out about one-quarter inch each 48 hours, for the last three dressings, with a return of the symptoms of generalized headache, eye pain, occasional vomiting and low fever. Drain of rubber catheter tubing reinserted under ether anesthesia, because of these symptoms. This drain was left *in situ* for ten days, then when discharge from epidural abscess lessened it was shortened each 48 hours until on May 24, when it was altogether withdrawn and the patient was discharged free from symptoms.

Summary: Recurrent mastoiditis. Protective meningitis; symptoms partially relieved by lumbar and cisternal punctures. Symptoms of petrous pyramid inflammation; unsuccessful exploration for epidural abscess. Right temporosphenoidal abscess; aspiration and drainage. Very severe symptoms of petrous pyramid inflammation, re-exploration for epidural abscess, localizing it, and drainage. Complete recovery.

140 East 54th Street.

ATYPICAL MASTOIDITIS. CASE REPORT.*

DR. HYMAN SPORN, Brooklyn.

In looking over the literature for the past 18 years, one is impressed with the fact that atypical mastoiditis is no longer apparently a rare condition. Especially during the past 10 years has there been an ever increasing amount of cases reported, showing that otologists are on the lookout for this condition. The condition occurs both in children and adults. In perusing the literature one is struck by the variety of names used to describe this condition, atypical mastoiditis, symptomless acute mastoiditis, mastoiditis without apparent infection of the middle ear, primary mastoiditis, idiopathic mastoid abscess, concealed or unobserved mastoid, etc.

McLoone, in commenting on this condition, states: "In mastoid surgery one occasionally meets with the most extensive pathological changes, notwithstanding the fact that the clinical symptoms did not appear alarming." One must realize that not all cases of acute suppurative mastoiditis are associated with symptoms. This is especially true for infants and young children. It is possible for the condition to be present together with dangerous complications, as perisinus abscess, labyrinthitis, extradural abscess, sinus thrombosis, meningitis, etc., yet the condition remains obscure because of the lack of any symptoms.

In the majority of cases of acute mastoiditis without complications, the signs and symptoms are such that the diagnosis is very readily made. The usual picture is as follows: a history of an acute rhinitis, shortly followed by otalgia, fever, insomnia, restlessness, spontaneous perforation of the tympanic membrane (or incision), otorrhea. Pain which may be present in the early stages of the disease becomes more severe. Tenderness and thickening of the mastoid periosteum usually is obtained. Deafness is noted early in the picture. Otoscopic picture shows a sagging of the posterior superior

*From the Dept. of Otolaryngology, Beth Moses Hospital, Brooklyn, N. Y.
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wall of the external canal. Leukocytosis is present and is of moderate amount. X-ray examination reveals a clouding of all structures, outlines indistinct with perhaps a suggestion or varying degree of breaking down of cell walls. It is those cases of mastoiditis, however, which have no discharge from the middle ear and are usually unassociated with the classical signs and symptoms of the disease that we are dealing with and which if not recognized may lead to serious and fatal complications.

The pathway of infection in atypical mastoiditis is variable. Trauma and blood stream infections are possible and can be passed over in naming. The usual explanation is that it is caused by an extension of the infection from the pharynx through the Eustachian tube into the middle ear. From here the process extends into the antrum and mastoid cells. Either due to a small aditus or swelling of the mucoperiosteum, the aditus becomes closed off. This gives rise to a closed cavity with conditions good for the progress of the infection. After the extension of the infection into the mastoid cells and the conversion into a closed cavity, the process in the middle ear and tube becomes quiescent. The symptoms of the process may be entirely lacking and our first inkling as to the nature of the process may be a subperiosteal abscess or a dangerous complication. Extension to other structures may occur by blood or lymph route directly or by erosion of contiguous bone.

The following case is of interest for several reasons: First, because of its history. Second, absence of changes in the membrana tympani and middle ear. Third, absence of pain and constitutional symptoms. Fourth, presence of a cortical perforation into membranous canal and sinus involvement.

Case Report: M. B., age 40 years, a chauffeur, was first seen Feb. 20, 1933, with the following history: In early part of January he had a slight discharge from left ear. This cleared up in about 10 days and he was symptom free until four days before he was first seen, when he developed pain in left ear, with swelling in front of and behind the ear. The pain was increased by movement of the ear. When he first entered office, he remarked to the nurse that he suddenly felt a discharge in ear and considerable relief from pain.

Examination showed some edema over left mastoid and zygomatic regions. There was a profuse, creamy, pussy dis-

charge coming from furuncles on floor and posterior canal wall. After wiping the canal dry, the drum membrane was easily visualized. This was of normal lustre and appearance, with all the landmarks visible. The external canal was considerably narrowed by the external otitis. There was no temperature. Hearing was slightly impaired. Weber lateralized to left. No history of tuberculosis or syphilis.

X-ray taken on Feb. 22, 1933, was essentially negative. Wassermann test was negative.

The discharge continued as profuse as at the beginning of the illness. Feb. 28 the left auricle became markedly indurated and reddened with few small vesicles present. The picture looked like an erysipeloid infection. There was no temperature. Under wet magnesium sulphate dressings, this condition cleared up in about four to five days. The ear picture at this time was about the same; external canal was narrowed by granulations through which pus discharged. Drum was of normal appearance.

March 11, patient developed two small preauricular abscesses. He was sent to hospital, where incision and drainage of these areas was instituted. Urine examination was negative. He was discharged in a week. Wounds healed in about ten days.

He was again seen on March 28. There was some postauricular edema unassociated with any pain. Never had any temperature. External canal was narrowed by granulations through which pus was seen exuding when postauricular pressure was made. Diagnosis of soft tissue cellulitis with a sinus into external canal was made. Conservatism and watchful waiting was advised in view of lack of any constitutional reaction.

On April 20, he complained of slight pain over mastoid. No temperature or insomnia. Examination revealed a small fluctuating mass the size of a hazel nut situated over the antrum region. He was again admitted to the hospital, where the following notes were made:

April 23: There is a soft tissue infection which is draining through left ear canal. For past four days has had slight tenderness over mastoid region. There is no temperature. X-ray taken yesterday is essentially negative. The postauricu-

lar edema extends over the zygomatic area. There is a small area of fluctuation postauricularly. Pressure over the antrum produces copious discharge of pus into left canal. The left ear canal is narrowed by granulation tissue on the floor and sides of the canal. The drum membrane is slightly thickened with all landmarks visible. Observation advised. Blood count: white cells 14,200 with 56 per cent polys.

April 24: Incision and drainage instituted of posterior small fluctuating area. Small amount of pus evacuated.

April 28: General condition good. There is a moderate discharge from postauricular wound and from external canal. Some postauricular edema still present. Middle ear picture still the same. In view of lack of constitutional symptoms to any appreciable degree further observation advised.

Picture remained the same until May 20, when another X-ray was taken. This revealed the following:

Right mastoid, well outlined and illuminated. Left mastoid, cloudy, cellular structure definitely indistinct with suggestions of breaking down. Impression, the suggestion of a low grade destructive mastoid is very strong.

He was again hospitalized May 28.

May 31: Methylene blue was injected into small postauricular wound. None observed entering external canal. No temperature. No tenderness.

Operation same day revealed a suppurative mastoiditis. Necrosis of mastoid process near zygoma and over canal wall, leading anteriorly to membranous portion of canal wall and posteriorly to a perisinus abscess. Periphlebitis extending from tip of mastoid to torcular end. Dural plate necrotic. Dura of normal appearance. All necrotic bone was removed, sinus was not touched. Iodoform packing.

Culture of left mastoid: Smear showed no bacteria, culture: staphylococcus albus and aureus. Excepting for first two days postoperative (100°), temperature before and after operation was normal. Patient discharged June 11.

Wound allowed to granulate in slowly. Discharge from external canal ceased on fifth day after operation and has

remained dry since. Granulations have markedly decreased. Drum at present is slightly thickened. All landmarks are visible.

CONCLUSIONS.

1. Extensive mastoid disease may occur without any alarming clinical or constitutional symptoms.
2. We must always be on guard for cases of atypical mastoiditis to avoid the danger of serious complications.

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- 762 St. Marks Avenue.

SUPPURATIVE PERFORATION OF LATERAL SINUS
WITH PREOPERATIVE AND OPERATIVE
HEMORRHAGE.*

DR. LOUIS R. EFFLER, Toledo.

The following case report offers some interesting findings. The patient was a woman, age 40 years. Whether this is just "one of those cases" or whether or not more practical conclusions can come from it beyond the closer observation and earlier operation of chronic suppurative mastoiditis, the reader is left to judge.

Past History: Patient had had some type of left mastoid operation at the age of 1 year. The wound had healed, but the left ear had been running continuously ever since.

Present History: Patient claimed to have contracted a "flu" about one week before our entry into the case. The net result of this infection was a flare-up of the left ear. Her complaints in brief included: a more profuse discharge of foul-smelling pus from the left ear; several attacks of actual bleeding from the left ear; severe pain in the left side of the head; and one or two attacks of chills and fever.

Physical Examination: Patient did not look particularly sick at this time. She was well developed and well nourished, conscious, and entirely rational.

Verification was quickly made of point 1 in her complaint. In support of point 2, a small amount of fresh clot but no actual frank bleeding was noted in the left ear canal. Cleansing of the latter revealed a completely destroyed drum and granulations covering the promontory. Tenderness was elicited over the left mastoid antrum.

Preoperative Diagnosis: Acute chronic mastoiditis, suppurative, left, with a suspicion of left lateral sinus thrombosis.

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This diagnosis seemed warranted by all the facts. Whether or not a "flu" was the factor that fanned a chronic into an acute condition or whether the acute stage was coincident with bacterial invasion of the blood stream were impossible to determine then or since.

Course: Patient was observed for the first twenty-four hours in the home. During this period, she had a sharp temperature rise to 104° , which was preceded by a half-hour chill. Patient was then transferred to hospital.

Her next three days in hospital developed several more sharp rises and falls of temperature. The highest point was 106° and the lowest 98.6° . Chills of greater or less intensity accompanied each rise. Her condition at this time looked serious.

A blood culture was made, but this eventually proved negative.

On several occasions fresh bleeding was noted from the left ear. This was rarely sufficient to do more than fill the ear canal, where it solidified into a clot and checked spontaneously. Proper evaluation of this bleeding was not made preoperatively.

Operation: On the fifth day, operation was performed. This was designed to include: a. radical mastoidectomy, left; b. inspection of the lateral sinus, left; and c. ligation of the jugular, left.

The usual curved incision was made over the left mastoid. The periosteum was stripped back in both directions. The cortex appeared to all intents normal. Before any effort was made to uncap the cortex and while the fibers of the sternomastoid were being stripped from the process, a sudden gush of blood obscured the field. Thought at first to be due to the postauricular artery or conceivably to an emissary vein, this hemorrhage assumed instantly alarming proportions. The field could not be dried even for a moment sufficiently to permit exploration. As a matter of fact, whole handfuls of sponges were so quickly saturated that it was necessary to use towels to aid in the control of the hemorrhage. Only one conclusion could be made, *viz.*, that this was a hemorrhage from the lateral sinus. Understand that the cortex had not even been uncapped. How the lateral sinus *could* be involved was then a mystery, but *that* it was involved seemed a certainty. The operating table and the operating floor soon

looked like a shambles. The fact that the bleeding seemed to come from the region of the tip availed nothing except as a point against which to apply pressure.

Many minutes elapsed before this violent hemorrhage was brought under control. Several attempts at inspection thereafter were defeated by a renewal of the violent bleeding. As a consequence, any further operative procedure on the mastoid was deemed absolutely impossible.

The jugular, however, in the left neck was quickly ligated. It was hoped that this step might at least prevent further infection from entering the blood stream.

Postoperative Course: On the first day, patient had a temperature of 105.8°, but with no chill. On the second day, she was irrational with either a secondary pneumonia or a pulmonary embolus in the lower left lobe. On the third day, she ranged from extreme restlessness to actual violence and suddenly expired in the midst of her struggles.

Autopsy Findings: The chief points of interest were:

1. *The Left Mastoid Tip:* It was easy to understand at autopsy why the operative hemorrhage was so difficult to control. It could not be attacked directly. The lower and the lateral aspects of the tip were completely destroyed. In fact, there was no cortex left at these points to guard the mastoid tip. It had been destroyed presumably by the long-standing suppurative process. All that remained was a ledge of bone that ended abruptly. A probe passed beneath this ledge revealed a large passage connecting directly with the body of the mastoid. The only structures holding back a lateral sinus flood before operation were soft tissues, *viz.*, periosteum and the interdigitating fibers of the sternomastoid. How much longer they could have succeeded in damming back this weakening dyke is impossible to say.

2. *The Mastoid Labyrinth:* This was found completely exenterated and filled with fresh blood and foul-smelling pus. The lateral sinus was partially exposed. Direct connections were established with the tract finding its exit at the tip and with another self-dissected tract communicating direct with the antrum. The former tract explained the severe operative hemorrhage and the latter tract explained the repeated pre-operative bleedings from the left ear canal.

3. *The Lateral Sinus:* A large perforation was found in the lateral sinus. From this perforation all the bleeding pre-operative and operative presumably took place. Pooling up in the self-dissected mastoid labyrinth, the blood *leaked* out into the ear canal and *poured* out at the tip.

Below the point of perforation the lateral sinus was grayish in appearance and brittle in character. A semi-organized thrombus completely obstructed its lumen. This thrombus extended below the knee and below the bulb, and involved the jugular in the left neck.

4. *The Jugular Vein:* The thrombus above described carried down into the jugular for a distance of about 2 inches above the point of its ligation. At this point it seemed to be more recent, softer and more lardaceous. Easily stripped from its bed, the vessel walls appeared roughened and apparently ulcerated.

5. *The Lungs:* The left lung showed two small abscesses (one-half inch and three-quarter inch in diameter, respectively). These were probably embolic in character. They explained the lung findings just before death. The rest of the autopsy was substantially negative.

SUMMARY.

a. A chronic suppurative mastoiditis is a serious menace to life.

b. A superimposed acute process may develop at any time. In this case the lapse was 39 years.

c. The chronic suppurative process in this case destroyed the whole mastoid labyrinth. In so doing, it also eroded the cortex at the tip, the cells at the antrum, and the lateral sinus wall.

d. The preoperative bleedings from the ear canal were lateral sinus leakages, though not recognized as such.

e. The operative hemorrhage from the tip was from the lateral sinus. It was recognized, not from its locality, but from its severity.

f. The cause of death was septicemia. The same perforation in the lateral sinus that permitted a leak of blood permitted an entry of organisms.

222 Michigan Street.

**PTERYGOMAXILLARY ABSCESS ASSOCIATED WITH
ACUTE SUPPURATIVE OTITIS MEDIA SIMULATING
ZYGOMATIC MASTOIDITIS. REPORT OF A CASE.**

DR. SAMUEL D. GREENFIELD, Brooklyn, N. Y.

Pterygomaxillary or infratemporal abscess assumes otologic importance when it is associated with a suppurative otitis media. Its signs and symptoms may simulate zygomatic mastoiditis so closely that one is apt to err in diagnosis with the unhappy result that the patient is subjected to unnecessary mastoid surgery. In order to obviate the possibility of such an error one must understand clearly these two clinical entities with special reference to the anatomy, pathology and symptomatology. It is with this thought in mind that I have undertaken this communication.

Pterygomaxillary abscess is a rare occurrence. Grant,¹ in a review of the literature found recorded only four cases of abscess of the pterygomaxillary space. Of these four cases, only two occurred in connection with otitic infection.^{2,3} Grant's case followed several weeks after the subsidence of an acute otitis media. My case developed intercurrently with the ear infection. This therefore makes a total of four cases of pterygomaxillary abscess associated with otitic suppuration that will have been recorded in the literature.

The pterygomaxillary fossa is that space bounded laterally by the ramus of the mandible, medially by the lateral wall of the pharynx and pterygoid process of the sphenoid bone, anteriorly by the zygomatic surface of the superior maxilla and superiorly by the lower surface of the greater wing of the sphenoid and the adjacent temporal bone. Posteriorly the space is limited by a line passing from the posterior border of the ramus of the mandible to the pharynx.

The pterygomaxillary fossa contains a number of structures, but for our discussion these of importance are, the chorda tympana nerve, a part of the parotid gland and the

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internal maxillary lymph nodes. Infection may gain access to this space from a number of sources, e.g., the middle ear, cells of the mastoid process, dental caries in the upper jaw and following injection of novocain in conductive anesthesia for tooth extraction.

According to Piersol, cases of otitic suppuration may propagate their infection from the middle ear to involve the parotid lymph glands. The latter drain the tympanum. These in turn communicate with the internal maxillary lymph nodes, which may break down and localize in the pterygomaxillary space. Grant suggests that another avenue from the middle ear cavity is created along the course of the chorda tympana nerve. The latter passes from the tympanum through the glaserian fissure to the infratemporal fossa.

In mastoid disease the process may proceed from diseased or ruptured cells situated in the digastric groove. Infection in these inner tip cells may point toward the pharynx and localize in the pterygomaxillary space. Infection may be carried by the lymphatics from carious teeth in the upper jaw. This is probably a frequent occurrence and is the source of infection in these cases not accompanied by otitic disease. The case reported by Foster⁴ falls into this category. His patient had no involvement of the ear.

Lastly we consider those cases in which infection results from the so-called mandibular injection by the dentist in the extraction of teeth. The mandibular foramen is used and very often some of the solution is directed into the pterygomaxillary fossa. It can readily be understood therefore that the needle may carry infection directly into this space. As an etiological factor this source of contamination must be reckoned as an important one. In the case I am recording this seems to have been the avenue through which the infection entered, even though there was an associated middle ear suppuration.

The signs and symptoms of pterygomaxillary abscess are fairly typical. The patient begins to complain of pain in the anterior auricular region and side of the face. This may radiate up to the eye, forehead and over the parietal region. There is some difficulty in opening the mouth, but this symptom does not become pronounced until later in the disease. Soon a swelling appears in the anterior auricular region,

which gradually increases in size until it extends up to the zygoma. It may spread around the auricle to involve the mastoid area. When the edema is at its maximum careful examination reveals the fact that the edema stops short of the zygoma. The maximum swelling therefore is below the bony arch. This observation is an important one in differentiating it from zygomatic mastoiditis. There is pain upon deep palpation over the swelling, but pressure over the root of the zygoma fails to elicit any tenderness. There is temperature varying between 99 and 103. Anorexia is present and the patient finds difficulty in taking food owing to the marked trismus. Suffering is intense and the patient appears very ill.

Examination of the upper jaw may reveal the presence of carious teeth or decayed roots. But more important than this finding is the elicitation of a recent dental history. The extraction of teeth under conductive anesthesia is very significant. Such a history coupled with the signs and symptoms above described makes the diagnosis of pterygomaxillary abscess certain even in the presence of a discharging ear.

The one condition that simulates pterygomaxillary abscess and from which it must be differentiated is zygomatic mastoiditis. That these conditions may resemble each other very closely is evidenced by the fact that all the otologists who saw my case as he sat in bed with a discharging ear, temperature and the zygomatic swelling immediately ventured the diagnosis of zygomatic mastoiditis. It was only after careful examination and especially after intimating the dental history that one suspected that this was not the correct diagnosis.

Zygomatic mastoiditis as we know is simply an extension of bone necrosis from the body of the mastoid into the zygomatic process of the temporal bone. Owing to the fact that the cortex is less dense at the base of the zygoma perforations most often take place in this location.⁵ The periostitis gives rise to edema over the root of the zygoma, which varies in extent in different cases. It may extend forward to the outer canthus of the eye, up over the squama and back over the mastoid region. A striking feature is that the maximum swelling is always found to be above the zygomatic arch. Pressure over the root of the zygoma always elicits exquisite tenderness. Trismus is never associated with zygomatic mastoiditis.

The following is a table showing the points of importance in the differential diagnosis between pterygomaxillary abscess and zygomatic mastoiditis:

Case Report: S. M., male, aged 15 years, was referred to me on February 7, 1933, the chief complaints being pain in the right ear, otorrhoea of three days duration and temperature.

Previous Personal History: Irrelevant except for the extraction of a right upper second molar four days before onset.

History of Present Illness: Six days prior to my first examination the patient had a severe head cold. Two days later he experienced pain in the right ear. His physician prescribed warm irrigations and by the next morning the drum ruptured and a profuse sero-sanguineous discharge appeared. The pain in the ear was not relieved after rupture of the drum and the patient was referred to me.

Examination February 7, 1933. General condition of the patient was good. Temperature at 11 A. M., 100-6. Mastoid was moderately tender over the tip, but there was absence of edema. Discharge was moderate and pulsation was visible. The drum was edematous, but no canal changes were noted. Hearing test showed whispered voice six feet from concha. At this time the patient presented an ordinary acute suppurative otitis media with some inflammation of the tip cells of the mastoid.

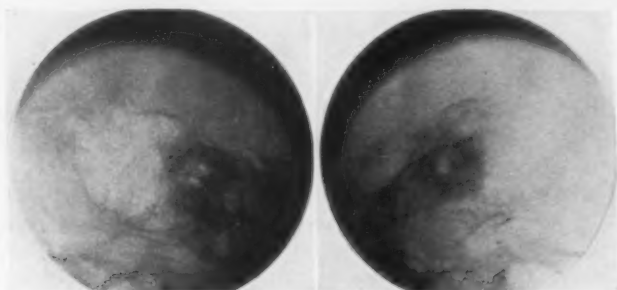
He continued irrigations at home for a period of a week. I saw him again on the eleventh and on the fifteenth. On the latter date the tenderness over the mastoid had entirely disappeared. The discharge from the middle ear was moderate in amount and the drum was flat. Whispered voice was heard eight feet distance. The patient complained of no pain, had no temperature and it seemed that the process was arrested and the ear was resolving.

The patient returned to me on the 19th. At this time his ear picture was unchanged. He complained of severe pain in front of the auricle and on the right side of the face. There was no visible swelling nor was there any tenderness. I advised the application of heat externally. I could not for the moment explain the cause of the pain.

	Preceding otitic infection.	Signs of existing mastoid disease.	Trismus.	Swelling over zygoma.	Tenderness.	Dental history.
Zygomatic Mastoiditis.	Always present.	Always present.	Never present.	Maximum swelling situated over or above the zygomatic arch.	Always present over the root of the zygoma.	Never present.
Pterygomaxillary Abscess.	May be present, but not necessarily so.	Usually not present.	Usually present to a marked degree.	Maximum swelling found to be below the arch of the zygoma.	Never present over the root of the zygoma, but only over swelling.	Very often present and of great significance.

Chart showing the differential diagnosis between Pterygomaxillary Abscess and Zygomatic Mastoiditis.

Two days later I noted a swelling on the side of his face immediately below the zygoma. Palpation disclosed some tenderness in this region and there was slight difficulty noted in opening the mouth. The swelling increased in size so that by the end of the week it involved the area about the zygoma and extended around the auricle to the mastoid region. There was definite pitting over the body of the mastoid as far back as the emissary. His ear continued to discharge and the temperature fluctuated between 99° and 103° . The pain was referred to the ear and radiated up to the eye. Firm pressure over the mastoid and especially over the root of the zygoma failed to elicit any pain.



Left Mastoid.

Right Mastoid.

Fig. 2. Shows the generalized clouding of the right mastoid.

The patient seemed to be in great distress. With swelling about the zygoma and a discharging middle ear, I thought I might be dealing with some atypical form of zygomatic mastoiditis. The patient was admitted to the Beth Moses Hospital on February 25, 1933, for further observation and study.

Admission Note: The patient is a young adult male apparently suffering severe pain. Temperature 102.4 , pulse 99, respirations 25. There is edema over the zygoma extending above and behind the auricle. Exquisite tenderness is noted over the parotid region and especially under the zygomatic arch. The aural discharge is profuse. There are no changes in the post-superior canal wall. However, there seems to be an antero-posterior encroachment on the canal lumen due to a bulging of the anterior wall. This area is not tender to the probe. There is marked trismus. The teeth can not be separated for

more than one-half inch. Fig. 1 shows the patient on the day of admission to the hospital.

Blood count, hemoglobin is 70 per cent, R.B.C. 4,000,000, W.B.C. 18,000, poly count 84 per cent. Urine examination, negative. X-ray of the mastoid disclose normal markings on the left. The right reveals generalized clouding of all the cells with some suspicious areas of beginning septal disintegration. Fig. 2 shows the X-ray plates at this time.

The patient was made comfortable with codein and heat was applied externally. The case was extremely interesting. A number of otologists saw the patient and the general opinion, especially when viewing the X-ray plates, was that we were dealing with a zygomatic mastoiditis. I could not become reconciled to this fact because of the following reasons: (1) The swelling was not typical of zygomatic mastoiditis. It seemed to stop short of the zygoma. In those cases of zygomatic mastoiditis I have seen the maximum swelling was above the zygoma. (2) There was absence of tenderness over the root of the zygoma. In all the cases of zygomatic mastoiditis I have seen exquisite tenderness was always found over this point. (3) The otoscopic picture showed a resolving drum and the hearing was unusually good for an operative mastoid with a four weeks history. (4) I could not satisfactorily explain the presence of the marked trismus on the basis of zygomatic disease. Theoretically, one might suspect extension of zygomatic disease to the temporomandibular articulation. I have never seen such a complication. (5) The dental history which dated back a few days before the onset of the otitis made me attach more importance to it than I would ordinarily have done in the face of these findings. For the above reasons I refrained from opening the mastoid.

On March 2nd, I decided to explore the pterygomaxillary fossa and at the same time inspect the bony zygoma for evidence of bone involvement.

Under gas-oxygen anesthesia I made an incision along the upper border of the zygoma. The bone was freed of temporal muscle fibres and inspected carefully back to the superior meatal wall. The surface of the bone was smooth and there was no evidence of a perforation or of disease. A long hemostat was directed downward under the zygomatic arch for a distance of one and a half inches where it entered an abscess

cavity in the pterygomaxillary fossa. Upon opening the hemostat an abundance of creamy pus began to exude. This was wiped away repeatedly and then the cavity was flushed with saline. A rubber tube was inserted and part of the incision was closed with silk-worm gut sutures.



Fig. 1.



Fig. 3.

Fig. 1. This shows the typical swelling of the tissues extending up to the zygomatic arch. The edema has also involved the mastoid region and the auricle is displaced forward and downward.

Fig. 3. This shows the line of incision over the superior border of the zygomatic arch. The wound is granulating and still discharging.

Soon after the operation the pain was relieved and within twenty-four hours the patient was free from symptoms. The temperature reached normal on the third day. The wound drained considerably and the rubber tube was retained in the cavity for one week. After its removal the wound began to granulate rapidly. The patient left the hospital on March 21st. Fig. 3 shows the patient on the day of discharge. The only remaining symptom was some limitation in opening the mouth. He was discharged as cured on April 12th, 1933.

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169 New York Avenue.

THE TUBERCULOUS LARYNX AND UPHILL FEEDING.*

DR. C. D. VAN WAGENEN, New York.

The tuberculous larynx has always been rebellious to the passage of nourishment. That this is so seems largely due to the location of an extremely sensitive infection within a semi-rigid surrounding. Feeding is so difficult and terminates so frequently in nasal regurgitation and prolonged, exhausting, spasmodic cough that those so afflicted prefer thirst and starvation to any repetition of this harrowing experience.

It seems possible to eliminate these two formidable obstacles through the use of uphill feeding. The process rests on two main factors. Not only must the larynx be desensitized, but there must be abolition of all reflex inhibitions arising from nose, nasopharynx and mouth, such as from deviated septa, postnasal discharge or sharp inner edges of teeth. The patient must be relaxed in bed and, particularly, all muscles of the neck and thorax must be divorced from the functions of support of the heavy head and maintenance of equilibrium, forming the semi-rigid surroundings and left free to devote themselves to breathing and swallowing.

To this end the patient is turned to the prone position in bed with head and neck projected beyond the side. This posture will place the mouth at a slightly lower level than the stomach. This posture also throws the larynx away from the esophagus, increasing the bypass space. A rectangular, resilient pillow is so placed under the thorax that it will not interfere with the arms nor encroach on diaphragmatic or abdominal movements, while permitting the chest to "bed itself down" to a comfortable pose. Some form of adjustable headrest is now brought up to the patient's forehead and the weight of the head gradually taken up until the patient seems and is entirely relaxed, breathing freely and quietly. The container with liquid nourishment is brought under the patient's face, preferably placed on a convenient stand, and instructions are given to drink, *slowly*, through a glass tube,

*Read before the New York Academy of Medicine, Section on Otolaryngology, May 17, 1933.

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steadied in the patient's hand. Once swallowing is started, siphonage contributes its aid. The situation is similar to swimming, where breathing and muscular action must effectually combine to produce rhythm. Breathing and swallow-

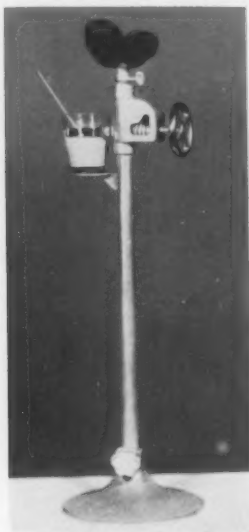


ing must coordinate. The first attempt will be timid and must be encouraged. Later, eagerness must be restrained else the whole process will become wrecked on the spasmodic cough.

The principle will achieve its greatest fulfillment in private practice. The degree of success will be in proportion to the confidence inspired and the intelligence of the patient. Because a considerable number of these unfortunates give a distinct alcoholic history, the combination of alcohol, in gradually increasing strength, with the anesthetic will be found of considerable ingratiating aid, aside from the cleansing and analgesic properties. "Some cocktail," has been the surprised comment on the initial use of this combination.

In intralaryngeal applications nothing seems to so completely anticipate spasm as the Laryngeal Medicator devised by the late Dr. Sidney Yankauer. Designed for self use, it may, later, permit the patient to participate in his own treatment, thus raising his morale.

The accompanying photograph illustrates the application of this principle to ward work. This apparatus, manufactured by the Kny-Scheerer Corporation, Long Island City, N. Y., is built to work at the side of a standard hospital bed and will be useful only at the bedside conforming to these standard measurements. But uphill feeding may be done at any bedside if the above noted directions are carried out. The principle is not remedial, per se, but humanitarian. Why stop at



this point in our efforts? In other situations we do not hesitate to fight with every means at our command for life, even though we know the termination will be fatal. Cautery, light, transfusion (many small doses) for the depressed bone marrow, etc., all can be pressed into service in our effort to ease the patient out of this life and mitigate the tragedy for family and friends. Once we have become emancipated from generations of hopeless prognostic teaching we may be better equipped to overcome the political inertia of urban concentrations and view the future from some better vantage point than mass segregation.

667 Madison Avenue.

A NEW SUBMUCOUS ELEVATOR.*

DR. ROMEO A. LUONGO, Philadelphia.

This is a modification of the Freer submucous elevator: It is narrower, thinner and bent to an angle on one end.

It was originally designed for the elevation of the mucosa from the lower (nasopharyngeal) surface of the sphenoid during the external frontoethmosphenoid operation; but it has been found very useful also for the elevation of the orbital periosteum in the beginning of the same operation, and for the elevation of the mucosa from the antrum and from the floor of the sphenoid during radical procedures.



Fig. 1. The submucous elevator.



Fig. 2. The suture-carrier knot-tier.

In performing a submucous resection I find it very useful for the initial elevation of the perichondrium and for getting around ridges and spurs that are so close to the nasal floor that there is not enough clearance to allow the use of the regular Freer elevator.

It is finished in black in order to prevent the annoying light-reflexes and to give a clearer view of the tip of the instrument against the white background.

THE SUTURE-CARRIER KNOT-TIER.

Ligation of the anterior ethmoidal artery can be done with this instrument alone. The catgut is threaded through the

*Read before the Philadelphia Laryngological Society, Nov. 7, 1933.

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small ring at the end of the instrument and then a knot is made about a quarter of an inch (0.6 cm.) from the end of the catgut (see Fig. 3C). When the catgut is stretched, the knot will become engaged into the ring and the stiffness of the catgut will keep the free end (beyond the knot) in such a position that it can be easily grasped with a small hemostat or forceps. The knotted end of the catgut is pulled out while the instrument is kept in place (the ring will protect the artery from the saw-like motion of the catgut against the

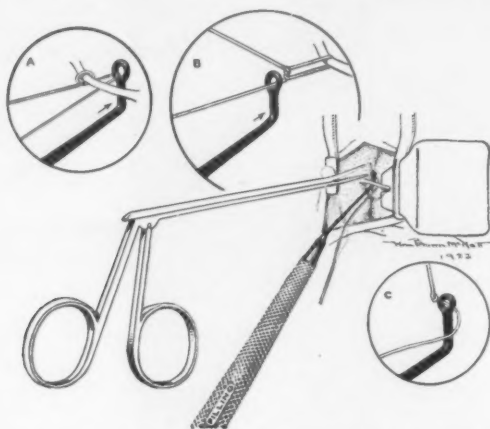


Fig. 3. The tying of the anterior ethmoidal artery with the suture-carrier knot tier. (C) represents the threaded instrument, showing the knot and the free end of the catgut above it (the knot will become engaged into the ring by pulling on the other end of the catgut). The central figure shows the instrument in place around the artery with the free end of the catgut (beyond the knot which is already engaged into the ring) ready to be grasped by a Hartmann alligator forceps. (B) represents the process of pushing the knot toward the artery. (A) represents the process of tightening the knot around the artery.

vessel during this pulling). Without being unthreaded, the instrument is then withdrawn, and a knot is made between the ring and the artery. This knot is pushed toward the artery and tightened as illustrated in A and B of Fig. 3. Subsequent knots are made and tied in the same manner.

This instrument can also be used as a probe for intranasal as well as extranasal work.

These instruments are made by the G. P. Pilling & Son Co., Philadelphia, Pa.

1713 Pine Street.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTO-LARYNGOLOGY.

Meeting of October 18, 1933.

Samuel J. Kopetzky, M.D., Chairman; Wallace Morrison, M.D., Secretary.

DR. SAMUEL J. KOPETZKY: I am extremely gratified that we have with us tonight, not only a distinguished guest from the west, but also a visitor from Cuba. I wish to present to the Section of Oto-Laryngology, Dr. Fernandez Soto, of Cuba.

An Obscure Case of Sinus Thrombosis with Unusual Complications. Dr. M. J. Gerstley (by invitation).

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

Case of Right Acute Mastoiditis with Lateral Sinus Thrombosis, Septicemia (beta-hemolytic streptococcus); Operation and Recovery. Dr. G. W. McAuliffe.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

Parapharyngitis Following Infection of Lower Jaw After Tooth Extraction. Dr. S. Knof.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

Cases of Jugular Thrombophlebitis Following Infections of Face and Throat. Dr. Jas. W. Babcock.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

Symposium: Oto-Laryngological Problems in Sepsis.

DR. SAMUEL J. KOPETZKY: Every one of these case presentations could be discussed. In arranging this program, however, it was thought that these cases could be best handled by an authoritative statement from authoritative sources, and that in the discussion the special features of these cases would dovetail in.

Dr. August L. Beck will open the symposium on Oto-Laryngological Problems in Sepsis by his paper on "Parapharyngeal Infections and Internal Jugular Thrombosis; Diagnosis and Treatment."

Parapharyngeal Infections and Internal Jugular Vein Thrombosis: Diagnosis and Treatment. Dr. A. L. Beck.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

DR. SAMUEL J. KOPETZKY: Now, gentlemen, I present a man whose writings and whose experience in sepsis, particularly the sepsis from the ear, is vast. He comes to present a newer viewpoint. He is trying to bring us to leave traditional paths. I present Dr. O. Jason Dixon of Kansas City.

Newer Conceptions in the Management of Septic Sinus Thrombosis. Dr. O. Jason Dixon.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

DR. SAMUEL J. KOPETZKY: We have just heard a man who presents his thesis on the lack of necessity for ligating the jugular vein, based on animal experimentation. He demonstrates the reformation of blood channels. He presents his argument for the retrograde thrombosis rather than progressive with the blood stream. He makes his point that the clinical evidence of recoveries and lack of recoveries based upon a certain procedure is not scientific. One would have to study the presentation that Dr. Dixon recently made before the American Otological, Rhinological and Laryngological Society, and go into the details which he has only sketched here, before the implications contained in his work can be fully appreciated. I consider that the Section is most fortunate in hearing this presentation and having it on record here. Dr. Dixon has presented his ideas as to the lack of necessity for ligation of the internal jugular vein.

The discussion now again, with just these few sketchy remarks, is brought back to the continuation of the evening's symposium. I hope that Dr. Dixon is willing to have a free discussion of his point of view. We will continue the symposium with the presentation by Dr. Ralph Almour.

Discussion of the Basis for the Selection of the Type of Procedure in Sinus Thrombosis. Dr. Ralph Almour.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

DR. SAMUEL J. KOPETZKY: Now the issue is made. The two points of view have been presented and the evening, of course, would not be complete while the scientists were discussing the pros and cons of this moot question if the main issue before the practicing otologist was not taken in hand. Therefore, it is most logical that this symposium should be completed by a presentation of the treatment of sepsis from the medical standpoint. I present a medical man of distinction, Dr. Marcus A. Rothschild.

Summation of Treatment of Sepsis from the Medical Standpoint. Dr. M. A. Rothschild.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

DISCUSSION.

DR. LEE M. HURD: Mr. Chairman, ladies and gentlemen: Dr. Beck, I think, has absolutely covered this subject from beginning to end. His anatomy is solid, his symptoms are solid, and certainly his surgery is solid.

May I bring out the fact that about fifty per cent of these cases, if not treated properly and early enough, die? As an illustration of this, not long ago I heard of a case in the western part of the state. A man had a case with swelling of the neck. The tonsils had been removed. He had nicked into the soft palate somewhere. He made a small incision in the neck and put in a gauze drain. Then he called a consultant from Buffalo; the consultant pulled out the drain, and out came all the blood from the internal jugular vein and the patient died. That patient probably would not have lost his life had the proper treatment been applied as Dr. Beck has advised.

The question in the neck is between acute simple adenitis and acute cellulitis, which is a fulminating condition. We are all familiar with the former. Most of them come from the tonsil. Take out the tonsil early. In the latter condition you may have pus which is localized, but usually not. Two most serious symptoms must be considered. If the process shuts down on the airway, something must be done and done quickly. The other thing is spreading either into the vein or down into the mediastinum. You must be one jump ahead of it. Dysphagia, locking of the jaws, etc., are characteristic symptoms.

As to submaxillary cellulitis, I have seen three cases following removal of wisdom teeth.

Now there are three things that I would say are absolutely essential in this problem. In the first place is decision, decision of the surgeon to operate and operate early; not stand around and watch. Next is courage to go ahead when you do operate and find the source of infection. Third, is the knowledge of how to do it. Knowledge of the anatomy is not enough. In these cases the neck is swollen and distorted, and nothing appears where it belongs. I have opened some internally. I do not think it is worth while. If you do reach the process there is a small opening which drains poorly and takes a long while to resolve.

Here is one interesting case: I saw a man one afternoon, with his neck swollen from the jaw right down to the clavicle, and extending from the midline to the lateral side of the neck. I took him into the hospital. He was breathing very badly. I injected the 2nd and 3rd cervicals posteriorly with the patient sitting up. Then I put him down, to a half reclining position, to operate and he stopped breathing. I immediately did an emergency tracheotomy, opened the neck and got pus. That night he developed emphysema around the wound which extended into the mediastinum, choked the heart, and he died. I have another case now in the hospital illustrative of this condition. A woman of 35 or 40 years had tonsillitis two weeks ago. Her temperature went up to about 102.5°, then gradually came down in three days. The doctor tells me that she had very few, if any, spots on the throat. She began to eat. She felt well, but he said she ought to stay in bed a few days more. A week ago yesterday I saw her. She had had then for 48 hours some swelling of the neck. I had her admitted to the hospital. On the outside the swelling had extended down in the submaxillary space within two inches of the clavicle. I injected novacaine and incised parallel to the jaw and about one inch below it, right into the sheath about the bifurcation, because all of these spaces center there. I went down to the sheath and found a large gland but no pus. I opened the sheath but found no pus, and as Dr. Beck advised, used my finger and went up into the pharyngomaxillary and submaxillary spaces, but found no pus. I went along the sheath with my finger, but found no pus. She was breathing badly and her pulse was irregular because of irritation of the vagus nerve. I put in packing, simply laying gauze in the wound. She has practically recovered. Her temperature was never over 101°. The neck swelling has gone down and there are no spots on the throat, which has been pale. Throat culture yields pure hemolytic streptococcus and the neck culture was pure hemolytic streptococcus with a few staphylococci. There has been no pus to date. Certain strains of streptococcus hemolyticus produce no pus. In another twenty-four hours the infection would have extended down into the mediastinum.

The whole story here is to have the decision, the courage and the knowledge of how to proceed with these cases.

DR. JOHN M. LORE: Mr. Chairman, ladies and gentlemen: I will limit my remarks to the first speaker of the evening so that the others can discuss Dr. Dixon's controversial subject. I think we can save time if I talk to the lantern slides.

I have been very much interested in this subject and have done much cadaver work and animal experimentation. The point of importance, in the practical application of that anatomy to the man who is doing local tonsil work, is this: If you inject to a depth of one-half inch or more, you get on the other side of the constrictor muscle. You deposit that fluid in the pharyngomaxillary space. In the cadaver we have used methylene blue in an undissected specimen and then cut down, using the incision Dr. Mosher recommends and hunted for the dye. In practically all cases we have been able to demonstrate the presence of the dye, not only in the pharyngomaxillary space, but in the carotid sheath. That demonstrates, I think, how infection can spread and involve very important structures.

We have been making some studies on cross-sections. This is a cross-section of the neck. The level is fairly high.

In this case we tried to simulate a retropharyngeal abscess, to study how the infection would spread. If you notice, here is the carotid sheath with the blood vessels, the pharyngeal wall and the prevertebral fascia. The lipiodol has had a tendency to spread back of the carotid sheath, as you see. That brings up the question of method of approach. Dr. Beck has mentioned the approach anterior to the sternocleidomastoid muscle. The end result is that he must swing around to get to the prevertebral fascia. Incidentally the specimen inside illustrates how lax the tissue is there, bounded anteriorly by the buccopharyngeal fascia and posteriorly by the prevertebral fascia. It is surprising the amount of fluid you can inject there and how quickly it disappears. To me the indications are in attempting surgery in that region to go back of the sternomastoid rather than anterior to it.

This is a drawing which shows the mylohyoid muscle and the floor it forms. In the consideration of Ludwig's angina, this is very important. Infection on top of that muscle will gravitate anteriorly, but will also spill over the back and down into the neck into the spaces that Dr. Beck mentioned. I have here a drawing to illustrate another view of that. These infections, particularly those arising from dental injections, if seen early enough, can and should be incised from the inside. Before it gets down here it must, of necessity, be present above the mylohyoid muscle unless the infection is so big as to fill the submaxillary space. I have had about 22 cases, most of them of dental origin, and most of them have been treated from the intraoral route. Past that stage, don't waste time with picayune operations inside the mouth because the material has spilled over into the submaxillary space. Later on the approach from the outside is of course indicated.

Another point I wish to bring out is the question of pus in the submaxillary gland. It will simulate a Ludwig's angina. As a matter of fact, I think it can become a Ludwig's. The point of differentiation to me is that slight pressure on the gland, in a normal gland, should produce clear fluid out of Wharton's duct. If you get some drops of pus it is fair to assume that the infection is in the gland itself and not in the submaxillary space. If you can establish that fact, the indications are clear as to what to do. Incise the capsule of the gland without paying much attention to the space above. Do not burrow up into the pharyngomaxillary fossa. In this way you may miss the pus.

I want to emphasize a point which I am sure Dr. Beck would have emphasized had time permitted, and that is infections inside the laryngeal box. As I attempted to point out before, we have the pyriform fossa, which becomes continuous with the esophagus. Infections of the pyriform fossa are limited externally and will not spill out, but will immediately deviate laterally so that the air-way is impaired. I have been able to show this on experimental animals.

We try to make it a rule, in periesophageal infections which spill outside, that the approach should be along the anterior border of the sternomastoid muscle, for the reason that if you invade that space which is continuous between the buccopharyngeal and the prevertebral fascia and there is still pus there, you will get a mediastinitis. I think this is a very important point to bear in mind.

There is one other point I would like to mention that I have learned from sad experience. When you have decided to make an incision in the skin, outline it with mercurochrome before you drape. The tissues are so swollen and indurated and distorted that if you drape first and then try to palpate and orient yourself, you will find yourself above the jaw rather than below it. Outline the incision first.

I will not discuss the other presentations. Thank you for the privilege of discussing.

DR. MARVIN F. JONES: Mr. Chairman, ladies and gentlemen: I will confine my remarks principally to Dr. Dixon's paper. We have listened tonight to a classical presentation presented by a perfect salesman who certainly is to be

thanked for coming this long distance to present his subject. He is more or less like Daniel entering the lion's den, and is to be congratulated for his courage. If I agreed with him I would not be following my own faith. We have an operation which has been a classic over a good many years. We have in this operation a definitely outlined procedure. In cases where there is doubt about the procedure to be followed or the medication to be ordered, there are a number of different cures and a number of different operative procedures, but where the way is perfectly definite a single procedure stands or a medication stands as being practical and satisfactory. I know of no procedure or no operation that has proved more universally successful in the hands of the average operator than our present accepted sinus and jugular operation.

There are one or two points which I would like to stress. In the first place, if I understand Dr. Dixon correctly, it is the practice to put gauze plugs inside the vein. Our procedure is somewhat different. We make our incision, carry it back as far as necessary to take out the clot, having placed iodoform gauze on each end of the exposed field outside the vein wall, not in the vein. The gauze on the bulbar end is removed after the incision has been made and if we get free bleeding we are sure there is no clot below. This plug is replaced and the upper plug is removed. If we get free bleeding we are pretty sure we have opened a vein in which there is no clot. Suppose the clot is there and is removed. The vein will fall back in position and the pressure is put on the outside of the vein, not on the inside. It seems logical to me that if a venous channel were to be reformed, it would as easily reform through this procedure as it would by plugging it with muscle.

Now regarding the fever, chills and the trouble from bleeding afterward, that is something I have not experienced if I have located the focus. During the dressing the plug can be loosened, and if there is the slightest indication of bleeding, it can be left alone. You can go back the next day or the day after. Obviously the dressing can be done without disturbing the inside of the vein, and so far as I can see, without disturbing the convalescence of the patient.

There is one thing about the ligation procedure which is perfectly definite. If there is a blood stream infection and you ligate the jugular, inside of twenty-four hours the blood stream infection has disappeared as far as cultural methods can demonstrate. That seems to be a pretty logical answer to ligation.

It is very difficult, after a man has come all this distance and shown us the courtesy of presenting his work, to disagree with him, but nevertheless I feel there are among us certain individuals who have done this operation who would rather take a direct route to the site we wish to reach, rather than an indirect route, although it may look more fascinating. You cannot overlook the fact that the clinical results are excellent. Technically this newer method is perfect, but the clinical results must be considered. After all we are out to cure patients, and by the standard methods, until something else has been proved definitely better.

There is one more point I would like to discuss, the "facial weakness" mentioned in two separate papers this evening. We have already digressed to the liver and spleen, and I hope I may be permitted to digress also. "Facial weakness" is an indefinite term, and it can be easily determined whether that weakness is central or peripheral. In the distribution of the facial nerve we have a bilateral source of innervation. From the cerebral cortex of both sides we have fibers coming through the corona radiata and the internal capsule. Part of the fibers go to the opposite side, but also a homolateral supply to the upper third of the facial nerve. The superior one-third of the facial nerve, therefore, has a double supply, one from the homolateral and the other from the contralateral cortex. The supply to the lower two-thirds is a single one from the contralateral side. Therefore, in central paresis or paralysis of the facial nerve, we have the lower two-thirds involved and a paresis of those two portions, but the function of the superior portion is preserved by the

bilateral innervation. In a peripheral facial paralysis we have a total loss of function, that is, all three branches are involved. In central paralysis only the lower two branches are involved. In the papers that are presented, if that point is brought out, instead of saying that the patient showed a "facial weakness," we would know whether the lesion was central or peripheral.

I want to thank Dr. Dixon for presenting such a beautiful and well worked out, let us call it theory, until it is a little more definitely established.

DR. SAMUEL J. KOPETZKY: Since the case presentations, unless the gentlemen have something particularly to say in constructive criticism of the presentations, were designed to form the framework of the papers of the evening, I doubt whether we need or should discuss them in detail at this late hour.

I hope that Dr. Dixon will close this discussion. He has traveled far to present his views before us tonight, and we are very grateful to him.

DR. O. JASON DIXON: There is a sort of double controversy here. Perhaps I have tackled this thing tonight in the wrong way. I think the way for me to go about this problem is to let the other man explain why he ligates. Certainly we do not stop infections by ligation. Where are we to ligate if we have a phlebitis, and what happens if we do, and what happens to the new thrombus that forms at the site of the ligated vein? In all of the discussion I have not heard this evening, granting that the patient has a septicemia or bacteremia, what happens to the new thrombus down in the neck closer to the heart. The point I want to emphasize particularly is the substitution of muscle for gauze. Gauze is a foreign body rejected by the body. Such foreign bodies are not accepted and are prone to infection. This muscle answers the job and prevents the repetition of the infection. Abdominal surgeons have learned long ago to leave things alone, not disturbing infected wounds after the operation.

Dr. Jones said something in regard to the position of the packing. Is he not putting a foreign body up under the inner table? We must consider this a phlebitis, as Dr. Almour brought out.

We could argue this question of ligation far into the night. Clinically I have never seen any difference in the postoperative recovery of the ligated or the non-ligated vein. We are always prone to resort to the knife if the patient does not do very well. I think sinus thrombophlebitis is a greatly overrated disease, particularly since we have brought in transfusions and better medical care. They will recover if they remain peripheral and there are no visceral complications. I think we cannot draw too strongly on clinical conclusions.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTO-LARYNGOLOGY.

(Meeting of November 15, 1933.)

Samuel J. Kopetzky, M.D., Chairman; Wallace Morrison, M.D., Secretary.

Clinical Presentation of the Improvement in the Surgical Repair of the Facial Nerve. Dr. Arthur B. Duel.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

The Rationale of Surgical Treatment for Ozena. Presentation of Cases Operated by the Author's Method. Dr. Alfred Wachsberger.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

DR. SAMUEL J. KOPETZKY: It now becomes my extreme pleasure and privilege to present to the Section of Oto-Laryngology of the Academy of Medicine one of the leading otologists of the Middle West. He is known to many of us as an earnest worker and a serious thinker, and has contributed much to this problem which concerns us tonight.

I introduce Dr. Harry L. Pollock of Chicago.

A Consideration of Genuine Ozena. Dr. Harvey L. Pollock.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

DISCUSSION.

DR. THOMAS J. HARRIS: Mr. Chairman, honored guest, Fellows of the Section: I extend to our guest on behalf of us all our very deep appreciation for the honor he has conferred on us by coming here tonight and giving us such an illuminating, such an able and such a fresh paper. The subject is a subject that has baffled investigators for a long time. He has brought to us new stuff that is highly interesting. He has told us many things tonight that most of us are entirely ignorant of in regard to work that has been done in the past ten or five years. Then he has in addition to that made this contribution to the practical side of the work, namely, the cure.

The paper was so complete, Mr. Chairman, that I can only hope to emphasize a few points. With most of what Dr. Pollock has said I heartily agree. The first point that did suggest itself to me was that I was not quite able to follow him in his statement of the frequency of cases of ozena. I know that a distance of shorter than eight hundred or nine hundred miles will make a difference in the frequency of a particular disease, and what they have in such numbers in Chicago it does not necessarily follow we have in New York. Certainly it is true in the experience of many of us in the past twenty-five years there has been a great lessening in the numbers of ozena cases which we see, not only in our offices, but in our hospitals. I remember twenty-five years ago at the Manhattan the night clinic was made up pretty largely of cases of ozena. We would have every night six or eight or more of them, young girls with rosy cheeks, the picture of health, but with this miserable affliction of the nose. They would come to us until they got tired of coming and then would drift off to someone else. Most of us know of the mental effect of the odor of the disease upon a sensitive individual, until suicide seems to be the only salvation to such patients as these. So that anything that can be done, as by the operations of Lautenschlager and Halle, and the work of the guest of the evening is most welcome.

The contributions that have been made to the etiology of this subject, the work that Costiniu and St. Popian are doing in regard to the possibility of transmission, are exceedingly interesting. I have seen certainly a few cases that fall in with this decidedly. I remember a family where four or five were afflicted and the father and mother. The third generation I do not know that I have ever seen. I was particularly interested in the work spoken of done by Fleischmann, and the views that were brought out that in all probability there was a trophoneurotic element and that the nasal ganglion might be in large measure at fault. It is just such contributions as those I think that arouse us all to do more ourselves.

Now in regard to the treatment, very rightly has the essayist used the word palliative. In all the irrigations, etc., for better or worse, it has been a question of palliation. They are better under treatment and as soon as the treatment is stopped they have gone back. So, when this principle was developed that the reduction of space would produce good results, a decided step forward was made. I have had no experience with the Halle operation. I was interested in seeing the two cases presented tonight and congratulate the surgeon on the results he has had there. Among other things, I was both surprised and delighted to see that nasal breathing was not completely cut down. The young woman told me that she could breathe well through the nose, as did the young man. The objection has been made, as the essayist has brought out, that the results are not permanent. The case of the surgeon, Dr. Wachsberger, is rather different. I congratulate him on the results he is getting.

In regard to the operation that has been described tonight on the septum and the injection of various materials, when it was first suggested we tried a number of cases with paraffin. They were one hundred per cent failures. The paraffin, as those of you who have tried it know, does not fit in well. The membranes in many of the cases were very thin and atrophic, and the paraffin sloughed out and that was the end of our results.

I have always felt that the operation of Lautenschlager and Halle was a very formidable operation. So that when this question of the ivory implant was described by the essayist, I felt we had something we could easily grasp. The period of time, ten years, he has been doing this and the number of cases he has done all bear out in that direction.

I think we want to remember in speaking of the prognosis that there is a certain lessening of the symptoms as one gets older. I think that it is true that the person at sixteen or twenty is apt to be much more a sufferer than at forty or forty-five, but nevertheless there are enough of these cases to require or prompt us to do whatever we can to relieve their extreme suffering and agony. When the doctor comes and tells us all he did tonight, how with a comparatively simple procedure and no after treatment the patient was healed in the course of a few days and he got such results as relieving the odor, cutting down the amount of secretion, with no crusts, and in other words restoring happiness and peace of mind to people who are so afflicted that their lives are such a sad sort, we can say very truly that we are under great obligation to the doctor for reporting this tonight.

DR. LEE M. HURD: Mr. Chairman, Dr. Pollock, ladies and gentlemen: I would like to ask Dr. Pollock one question. It is a fact that the septum is usually somewhat deviated. Why didn't you take out enough of the septum to put the ivory splint in against the mucous membrane of the other side?

The points to keep in mind while seeking the cause are that most of them were females, most of them, as far as you can observe them, start at puberty and usually they go through to the menopause and then improve, and the symptoms subside to some extent. As far as I can remember, most of the cases I have seen were in Semitics. They came from poor sections and from Southern Europe, as Dr. Pollock said. It was a social status affair, like tuberculosis. They were poor people. I do not think I ever saw an atrophic

rhinitis in a person who had means, the food, surroundings and hygiene that they ought to have. As Dr. Harris said, we do not see so many in the clinics now as we did. I think it is because we have restricted immigration, and the next generation in this country are getting better conditions in life. I think for that reason they are not having this disease. I know that when I worked at Bellevue we had lots of them. They came from the great East Side. At the Polyclinic we rarely saw them, and saw none at all in private practice. I have seen one unilateral case this year in a woman with a badly deviated septum, with crusts on the concave side, and I saw one case last year of atrophy from extensive pansinusitis.

At to treatment, I will try Dr. Pollock's method if I ever have another one of these cases. It sounds very simple and much better than breaking into the lateral wall.

When the Bulgar bacillus came into popularity I tried it, using the living bacilli. They stopped the odor, but to get the living bugs for the patient was difficult and when they were put on the market for other purposes they were expensive. Then I searched for a cheaper substitute for the living bacteria, and let them buy Bulgarian milk in the drug store. They injected the nose with a small syringe, then an ordinary saline douche to remove the crusts and killed the odor. Long before that I was using citric acid as the mildest acid I could find in a snuff with sugar of milk. This would stop the odor, but not the crusts, because it made it dryer. I used one part citric acid to four parts of sugar of milk. As far back as twenty years ago I tried to get together and keep track of a number of children whom I discovered with atrophic rhinitis. Out of that number I have followed just one to date. That girl, then twelve years of age, came to me in 1913 with crusts, odor, headaches and all the picture of atrophic rhinitis. I treated her with iodine locally and Bulgarian milk. At the end of the first year she still had crusts. At the end of the second year the crusts had ceased, but the nose was full of thick mucus material. At the end of the third year the turbinates began to fill out. At the end of the fifth year she still complained of headaches, but no crusts and the turbinates were normal. Now after twenty years she has a normal nose, and this summer she came to me with hay fever.

DR. DUNCAN MACPHERSON: I want to express my appreciation to the speakers this evening. Certainly the very comprehensive paper by Dr. Pollock covered the situation very thoroughly. A good deal has been said about palliative treatment, and nothing about preventive treatment.

I agree with those who say that ozena is relatively rare compared to what it was some years ago. I do not think I have seen two cases in the last year which I would characterize as falling into the category we used to call ozena. I think there are a good many men in this room who would bear me out in stating that in this city we do not see many cases of ozena. What we call paranasal sinusitis used to be referred to as purulent rhinitis, especially in children. We do not hear anything about purulent rhinitis today. It is now sinusitis in children. It is, in a large majority of cases, curable by removal of the tonsils and adenoids, according to Dean, of St. Louis. He claims that eighty per cent of sinusitis in children is curable by removal of the tonsils and adenoids. That being the case, we do not have sinusitis today as we did before the removal of adenoids and tonsils became so common a procedure. We do not have atrophic rhinitis today as a sequel of sinusitis to the former degree, and we do not have so-called ozena, which is a type of atrophic rhinitis, to the same degree. Now in Southern Europe, where so many of these cases of ozena are found, I understand that operation for tonsils and adenoids has not reached anything like the stage of investigation that we have reached in this country. They do not attend to them any more than they attend to their teeth. I think that may cause a good deal of the ozenas they have over there.

We all feel that we have families afflicted with sinus disease, and so we have families afflicted with ozena. In many of these people sinus disease was

originally due to tonsils and adenoids. We removed them and today in these people operated on twenty or thirty years ago we do not see ozena, or very few cases. Like many other discoveries, the detection was incidental and accidental, and now with improvement in diet the results will be still better. With some discrimination, New York has for thirty years been attentive to adenoidectomy.

DR. HENRY H. BEINFELD: I have had occasion to treat a case of ozena recently and it might be interesting to follow out the medical treatment that I have used in this particular case. Considering ozena as an atrophic rhinitis I felt it was detrimental to use any form of irrigation which would increase the dryness. In this particular patient I used argyrol tampons to loosen the crusts. After that was cleaned I proceeded with gentle massage of the turbinates, using five per cent scarlet red on an applicator. After one and a half years' time the patient was rid of the odor and the crusts, was feeling very much more comfortable and was apparently perfectly cured. I wondered whether Dr. Pollock had used that method of treatment very much.

In line with what Dr. Hurd mentioned about taking out part of the septum, I thought it would be a good idea in these case of bilateral ozena to remove part of the septum and hold the ivory implant in place by making a groove on the under surface of the ivory so that it would rest on the cartilage which was partly removed instead of putting it in each side separately.

DR. ALFRED WACHSBERGER: I want to thank Dr. Pollock and the other speakers for their kind reception of my paper on my modification of the Lautenschlager operation. Certainly there is nothing to add to Dr. Pollock's exhaustive dissertation. There is something, however, which has to be emphasized, namely, that ozena should be attacked before it has progressed too far. One of the characteristics of ozena, as also of otosclerosis, is that it is progressive. A great step forward is made by treating the patient surgically and it should be done as soon as the diagnosis of genuine ozena is established. One of the patients, the youngest, was done under general anesthesia with as good a result as with local. I try to operate as early as possible.

In regard to the stuffiness, or fear of stuffiness, in the nose increasing in these patients by narrowing the nose, we find just the opposite to be true. They have stuffy breathing before, and after the operation they claim that they breathe more freely. I think this can be explained by the fact that the patient with the wide, dry nose does not realize that he is breathing through the nose, while after operation the dryness is gone and with a narrow, moist nose, he does realize it. Therefore, there is really no danger of stuffiness.

DR. V. J. ORLANDO: Dr. Pollock said something about palliative treatment. I wish in his secondary remarks he would tell us what his conception of palliative treatment is.

Before I sit down I would like to say that at the present time I have a young girl, sixteen years old, from a very well to do family, with what I believe is an incipient case of ozena. She is the picture of health. I do not agree with this contention that ozena occurs only in the poorly nourished. In my years of experience, both in private practice and in the various hospitals and dispensaries with which I have been connected, I have seen it in quite healthy individuals. That there probably is some hereditary predisposition I quite agree, as in some of my cases one of the parents had also had the disease. I also have seen it in more than one member of a family.

DR. S. L. RUSKIN: I would like to ask Dr. Pollock what were his observations on the pathology of ozena, whether he could differentiate the pathology of ozena from atrophic rhinitis as I have described it, and, whether his pathological findings showed endarteritis obliterans for ozena and round cell infiltration for atrophic rhinitis. These observations have been checked up in several institutions. I would also like to bring up the problem of whether

surgery is indicated in cases where the arteries are obliterated, because narrowing of the nose in these cases may increase the difficulty. Rather you must look to the source of the disturbance, whether it is vascular or infective structure. We all know we have cases of ozena and atrophic rhinitis with a considerable space in the nose, very little discharge and no crusting, and the mechanical factor is not a factor in the symptomatology. I would like to hear from Dr. Pollock about this. The disease is subjected to such extensive surgery as the Lautenschlager and Halle operations with results that are highly questionable that in the absence of more careful scrutiny of the underlying pathology no radical surgery should be advised. At the recent congress in Madrid, where I spoke on ozena, Halle himself told me he would not permit surgery on a nose with endarteritis. How can this differentiation of ozena from atrophic rhinitis be overlooked here when it is so fundamental to a rational approach to the subject? How can it be ignored in such an extensive discussion of this subject as this tonight?

DR. HARRY L. POLLOCK: Concerning the question of removing part of the septum, I would say that I have never tried it, never having had any occasion to use this method, but I suppose it would do no harm. When a bilateral ivory implant is done, in course of time the cartilaginous part of the septum disappears.

I fully agree with what Dr. Hurd said about these cases. As stated before, most of them are found in natives of or descendants of natives of Southern and Southeastern Europe, but we do occasionally find them in natives of Ireland and in the upper strata of society. However, they most frequently come from poorer families where the home environment is not the best. I also agree with Dr. Harris in that ozena cases are infrequently seen in the last two or three years. As previously stated, there is a predisposition present, but there must be an exciting cause, such as sinusitis or some infectious disease to bring about the presence of ozena. I think that the removal of tonsils and adenoids is a factor in relation to sinusitis, that by such removal we do eliminate many cases of sinusitis. Furthermore, we doubtlessly see fewer cases of sinus disease in recent years due to the fact that the majority of people take care of acute colds immediately and do not permit them to run along during the winter months without consulting a rhinologist.

I want to congratulate Dr. Wachsberger on the results of the three cases which he presented, as the spaces were narrowed and there was practically no crusting nor odor. As stated in my paper, I have not had any experience with his method, but it is so far removed from the Lautenschlager operation that it is hardly to be compared with it.

In answer to the question about palliative treatment, everything is palliative except surgery. This includes suction, irrigations, sugar of milk, iodine or anything else. Many cases of spontaneous cures have been reported in the literature. Occasionally some of these patients get well no matter what is used.

In regard to the last question, I stated in my paper that occasionally endarteritis obliterans has been found, but in the great majority of pathological specimens reported, very few cases of endarteritis obliterans have been found. Even in the event that such pathological condition is present, I still feel that the treatment indicated is the same—narrowing the space, which gives a physiological cure. What I mean by physiological cure was also here stated, namely: no crust nor odor and only an occasional mucoid discharge. I have had many cases following operation in which the nose was absolutely dry for many years, with discharge present only when an acute cold developed.

DR. SAMUEL J. KOPETZKY: In closing this discussion, I may say I have not observed the ivory implants. We know Dr. Pollock, we know his standing and we accept his results. On the other hand, when a young man like Dr. Wachsberger comes here by invitation to present his work, if one of us

of the senior group can give testimony to having seen his work, it will add to the record. I am glad to do so. I have seen his work in the hospital and watched the after results. I saw no particular untoward reactions and in the usual course of things there was a physiological cure, the types of which have been presented here. I am putting my statement and testimony on record to complete the records.

DISCUSSION OF PAPER PRESENTED BY DR. DUEL.

DR. MARVIN F. JONES: Mr. Chairman, it is a terrible anti-climax for me to say anything after a presentation such as the one you have listened to this evening. It is undoubtedly the outstanding piece of work of our generation, and I only wish there were some of us in the room who after a hundred years could leave such a monument behind us. The only excuse for my saying anything is perhaps from the point of view of a neophyte. Probably some of us will try this method. I have tried it in one case, and the mistakes of the neophyte will be helpful to others. My case was one which had gone four years since the injury and the nerve did regain its function, but the muscle did not regain its function. So, he has a partial improvement, but not the beautiful results that have been shown by the master, and I say that reverently tonight.

One of the rather ludicrous mistakes I made in my case was that I did not know the difference between gold foil and gold foil. There is the ordinary gold foil, and the ordinary gold foil is not the kind to use. If you have ever tried to handle it you know you are about like the monkey who couldn't eat the banana. It gets all over everything and you can't get it off of anything. Dental gold foil is the kind to use.

Incidentally, if you care to have some fun, go to the dead-house and do as many operations as Dr. Duel did on the cadaver and on the monkey, which incidentally is in itself a good point. As he tells it here, you will find that a number of things that sound simple are not so simple. I saw one of the cases he did. He wound up that afternoon about eight o'clock at night, and he and everyone else was ready to drop from exhaustion. That was the second case he showed tonight. He used the nerve of Bell and had some difficulty in finding it. However, in the dead-house, if you start to do these, there are two or three places that are troublesome. For those of us who do make mistakes, it is well to emphasize where they are made. In the first place, it is difficult to find the nerve. You will be surprised at the depth of the nerve when you try to find it, and you will be surprised to find how far anterior it is, and how much bone you have to take off in front of the tip. When approaching the nerve itself you will find a funnel arrangement that leads to the stylomastoid opening. When you are working in between the horizontal canal and the round window, you are in a delicate position. The nerve runs in a very narrow channel and it is difficult to get it out without dislocating the stapes.

It is not the simple process you would naturally suppose. The part that sounds the most difficult I found to be the easiest; that is, going along the inner wall where the nerve runs to the geniculate ganglion.

I wish I had the command of the English language or any other language to express my appreciation to Dr. Duel for this beautiful piece of work.

DR. SAMUEL J. KOPETZKY: Gentlemen, I think I voice the sentiments of this Section in expressing to both the visiting guest, Dr. Pollock, and our other speaker, Dr. Duel, the appreciation of the members of the Section. I thank you, gentlemen, for your contributions.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTO-LARYNGOLOGY.

Meeting of December 20, 1933.

Samuel J. Kopetzky, M.D., Chairman; Wallace Morrison, M.D., Secretary.

Osteomyelitis of the Skull. Dr. Charles C. Wolcott.

I have for presentation two cases of osteomyelitis of the skull. The first one is a little girl about one and a half years of age. When first seen she had orbital cellulitis (right) secondary to osteomyelitis of the right maxilla which had become infected from acute sinusitis. There was an erosion of the hard palate in the region of the floor of the antrum through which the antrum was cleaned. Thus no tooth buds were disturbed. One month later she developed an osteomyelitis of the frontal bone on the left side, contralateral to the original focus. This responded to surgery and transfusion. Six weeks later she developed an area of osteomyelitis at the junction of the occipital and parietal sutures. This responded readily to surgery. Some weeks later she fell down six stairs, fracturing her left parietal bone from the surgical defect posteriorly for three inches. She recovered from this without consequences. All lesions have healed and she has only a partial paralysis of the external rectus and OMPC, right. (Demonstration of slides.)

The second case is that of an adult who developed osteomyelitis of the right frontal bone secondary to acute frontal sinusitis and orbital cellulitis. He had a very stormy illness, requiring the removal of considerable bone. Subsequently he developed an abscess of the right frontal lobe which responded to drainage. He is well now and back on duty. (Demonstration of slides.)

A Case of Cranial Osteomyelitis. Dr. Hampton P. Howell (by invitation).

This young man, E. Z., alias "Jimmie," of Greek parentage, was admitted to our department of Roosevelt Hospital on June 14, 1933.

His complaint was severe pain and swelling about the right eye and root of the nose, at first accompanied by chilly feelings and followed by fever. There had been no history of cold in the head, and although he stated that he had been swimming five days before, he apparently had suffered no evil effects.

On examination, we found a well developed boy of about 18 years, with his right eye closed by a greatly swollen upper lid, accompanied by some edema extending above the rim of the orbit. There was some exophthalmos, with slight limitation of movement of the eyeball. The vision and appearance of the eyeball were normal, but there was marked chemosis of the conjunctiva above and below. There was no disturbance of eye grounds and nasal examination revealed no pus in the right naris, only some swelling of the mucous membrane high up in the vault of the nose. X-ray studies showed generalized haziness of the right antrum and ethmoid cells on the same side. The temperature on admission was 102.2°.

On June 16th, an ethmoidfrontal operation, under ether anesthesia, was made through a Killian incision. The ethmoid cells were greatly congested and contained a few drops of pus; on the other hand, when opened, the frontal sinus was found to be absolutely normal and, while there was some swelling of the intraorbital tissues, no real exudate was present. The wound was packed with gauze. Afterward the culture was found to be *staphylococcus aureus*.

The following day the wound was reopened in an attempt to institute better drainage of the orbital cavity, but on making a tract as deep as possible, only

inflammatory tissue was encountered. However, the internal orbital periosteum seemed to be greatly thickened. The temperature became septic in type, spiking from 104.5° down to 99°. A blood culture on June 25th was positive for staphylococcus aureus. A transfusion of 500 c.c. of whole blood was given on this day. Another transfusion of 300 c.c. was given five days later and repeated seven days afterward.

On July 1st, or two weeks from the commencement of the illness, it was noticed that the patient could not grasp articles with his left hand. Complete aphasia followed and he could co-operate but little during examination. Lumbar puncture subsequently showed clear sterile fluid under 350 m.m. pressure. Personality changes became evident and examination elicited several fluctuant areas over the right frontal region. Five weeks after the onset of the malady the boy's films showed an extensive osteomyelitis of the right half of the frontal bone with destruction of the posterior wall of the frontal sinus.

Realizing the rapidly progressive type of the bone disease and suspecting intracranial complications also, we transferred "Jimmie" to the surgical department, where he was put under the care of Dr. Howard Patterson, who will continue the progress to date of this most interesting and unusual case.

A Case of Cranial Osteomyelitis (Continued). Dr. Howard A. Patterson.

Dr. Howell has outlined the story of the first month of this boy's illness, prior to his transference to the general surgical service. Before presenting the patient, who is here this evening, I should like to outline very briefly the main features of this rather unusual case and to show a few lantern slides which tell the story far better than I can.

When this boy was transferred to our service there were two presenting problems, a rapidly spreading osteomyelitis of the skull and an obvious intracranial complication. In view of the recent positive blood cultures and the multiplicity of the neurological signs, it seemed likely that multiple brain abscesses were present.

On July 20, 1933, a very conservative right frontal procedure was done, with removal of a two-inch circle of bone in the area of greatest involvement. No fistulous tract leading through the dura was discovered, nor any other evidence of a frontal abscess.

Following this procedure the osteomyelitic process did not spread any further. However, the boy's condition grew much worse and he developed a paralysis of the left face, arm and leg. The optic discs showed an advanced degree of choking. There was marked left astereognosis. Finally he was unable to co-operate at all with the examination and lapsed into coma. Obviously we had to make an effort to drain the abscess (or abscesses), but the neurologists could not help us much with the matter of localization. It was decided to make a right posterior burr hole, the plan being to go through the bone furthest removed from the osteomyelitis, and to do a ventriculogram if the abscess was not encountered. At a distance of only 1 cm. from the dura, a large collection of pus was encountered. This was drained by aspiration and the scalp wound closed tightly without drainage. In a few hours all the paralysis, due obviously to edema, had disappeared. The abscess cavity at its height contained 300 c.c. of pus. It has been emptied by aspiration four times. On the last aspiration, six weeks ago, the capacity was only 45 c.c., and the contents were sterile. All previous taps had shown staphylococcus aureus. There is some doubt whether the large abscess was really intracerebral. It certainly was intradural, and I feel that its shape as shown in the slides, is in favor of an actual intracerebral location. The lantern slides, with the cavity outlined by air following emptying of pus, are very striking.

The boy's general condition has improved amazingly. He has regained 40 pounds of his lost weight. His vision is normal. For the past three and a half weeks his temperature has been low, normal most of the time, but showing a slight rise about every third day.

The Regression Theory of Otosclerosis. Dr. L. K. Guggenheim.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

DISCUSSION.

DR. SAMUEL J. KOPETZKY: I am sure this Section has rarely had the pleasure of hearing a more scholarly address upon an original topic than the one we have just heard. Our own member, Dr. Duel, who has worked and thought a great deal upon the subject of otosclerosis, in addition to his other work, will open the discussion.

DR. ARTHUR B. DUEL: Mr. Chairman and gentlemen: I am sure we are all very grateful to Dr. Guggenheim for coming all the way from St. Louis to expound his philosophical study of otosclerosis. It is always more convincing when a philosopher backs up his sage remarks by such material proofs as he has shown us here tonight. Dr. Guggenheim presents his arguments so charmingly that I always believe him even if I do not understand what he is talking about!

As early as twenty-five years ago I opened the discussion of a paper by Dr. George Shambaugh on otosclerosis. I have brought with me a copy of the *Transactions of the American Otological Society* for 1908. At that time, while there were a few who thought of the possibility of this being an hereditary disease, it was far from a generally accepted idea. Many men who had given the matter most careful thought felt that there were other factors in the production of otosclerosis. At that time, as I was going to this meeting, I remember speculating on how I should approach the discussion. I hardly dare to call myself a philosopher on this subject after listening to such a master of philosophy as has spoken to us tonight. I had better qualify myself as a "pseudo-philosopher." At any rate, I determined to emphasize, in the discussion, the idea that otosclerosis was probably hereditary. Just at that time there had appeared a very popular paper in *Harper's Magazine* expounding the Mendelian idea of heredity. I read a little resume of this article; a very simple explanation of Mendel's findings which had been made way back in 1867, and which had remained dormant until about 1897. Then, in the subsequent ten years, it had been verified and the principles were being used in animal breeding and horticulture. At the end of that discussion I declared the belief that if anything really was to be done for otosclerosis, looking at it from the practical clinical point of view, it would have to be done through genetics. In other words, we would have to breed it out if we expected to wipe it out.

Many years later, at the opening of the International Oto-Laryngological and Rhinological Congress in Copenhagen, I read a paper on "The Outlook for the Solution of the Problem of Otosclerosis," in which I was still interested. Again quoting, I said: "So far as I can discover, nothing but eugenic prophylaxis can possibly be of any service at present." I suggested a plan of correlated research in which a continued study of the end result would be replaced by a study of etiology.

The very important thing to me about this genetic idea is that otosclerosis, or otospongiosis, or really a spongiosis of the petrous bone, is not, per se, an ear disease. It is, rather, a disease of the temporal bone which *sometimes causes deafness*. When it causes deafness it is because this disease happens to take place in a bone in which the very important end organ of hearing is placed. Now the only method, in my opinion, by which we will advance in an understanding of otosclerosis lies in the study of potential cases in which genes of otosclerosis may be present. Embryos, or still-born children, or children up to 2 years of age, are the ones who are going to answer the problem. This is a disease of the petrous temporal bone (and probably of other bones), which may eventually be found to be as common as tuberculosis. In a few instances it mechanically produces deafness.

(To be continued.)

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